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Pulmonary Barotrauma Resulting from Mechanical Ventilation in 2 Patients with a Diagnosis of COVID-19 Pneumonia

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ADEF 1 Raphael Ezeagu
AEF 2 Titilope Olanipekun
F 1 Ratnam Santoshi
E 1 Chanaka Seneviratne
E 1 Yizhak Kupfer

1 Department of Pulmonary and Critical Care, Maimonides Medical Center, Brooklyn, NY, U.S.A.
2 Department of Hospital Medicine, Covenant Health System, Knoxville, TN, U.S.A.

Corresponding Author: Raphael Ezeagu, e-mail: raphaelezeagu@yahoo.com
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Case series
Patients: Male, 71-year-old • Female, 58-year-old
Final Diagnosis: Pulmonary barotrauma
Symptoms: Pneumomediastinum
Medication: —
Clinical Procedure: Chest tube
Specialty: Critical Care Medicine

Objective: Unusual clinical course

Background: Invasive mechanical ventilation can cause pulmonary barotrauma due to elevated transpulmonary pressure and alveolar rupture. A significant proportion of COVID-19 patients with acute respiratory distress syndrome (ARDS) will require mechanical ventilation. We present 2 interesting cases that demonstrate the possibility of COVID-19-associated ARDS manifesting with pulmonary barotrauma at acceptable ventilatory pressures.

Case Reports: The first patient was a 71-year-old man who was intubated and placed on mechanical ventilation due to hypoxemic respiratory failure from SARS-CoV-2 infection. His partial pressure of O2 to fraction of inspired oxygen ratio (PaO2/FiO2) was 156. He developed subcutaneous emphysema (SE) and pneumomediastinum on day 5 of mechanical ventilation at ventilatory settings of positive end-expiratory pressure (PEEP) ≤15 cmH_2O, plateau pressure (Pplat) ≤25 cmH_2O and pulmonary inspiratory pressure (PIP) ≤30 cmH_2O. He was managed with 'blow-hole' incisions, with subsequent clinical resolution of subcutaneous emphysema. The second patient was a 58-year-old woman who was also mechanically ventilated due to hypoxemic respiratory failure from COVID-19, with PaO2/FiO2 of 81. She developed extensive SE with pneumomediastinum and pneumothorax while on mechanical ventilation settings PEEP 13 cmH_2O and PIP 28 cmH_2O, Pplat 18 cmH_2O, and FiO2 90%. SE was managed with blow-hole incisions and pneumothorax with chest tube.

Conclusions: Clinicians should be aware of pulmonary barotrauma as a possible complication of COVID-19 pulmonary disease, even at low ventilatory pressures.

MeSH Keywords: Barotrauma • COVID-19 • Emphysema • Respiratory Distress Syndrome, Adult

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/927954

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Background

Invasive mechanical ventilation can cause lung injury from pulmonary barotrauma and regional lung overdistension [1]. Pulmonary barotrauma leads to air leaks into the extra-alveolar tissue, causing subcutaneous emphysema, pneumothorax, pneumomediastinum, pneumopericardium and pneumoperitoneum [2,3].

Pulmonary barotrauma from mechanical ventilation usually occurs in the setting of high ventilatory pressures: peak inspiratory pressures (PIP), plateau pressure (Pplat), and positive end-expiratory pressure (PEEP). High PEEP is essential to maintaining lung recruitment while preventing atelectrauma [4]. Studies have shown that patients with underlying chronic lung diseases requiring invasive mechanical ventilation, such as interstitial lung disease, emphysema, and asthma, are more likely to develop barotrauma [5,6]. Notably, acute respiratory distress syndrome (ARDS) as both a primary indication for mechanical ventilation and complication during ventilation has also been identified as an independent risk factor for barotrauma [5–7]. Even in the absence of mechanical ventilation, patients with ARDS are at risk of patient self-inflicted lung injury (P-SILI) from an increased ratio of dead space to tidal volume during spontaneous breathing [8].

Currently, there are more than 1 million cases and 80 000 deaths attributed to the COVID-19 infection pandemic worldwide [9]. The virus that causes COVID-19 is designated SARS-CoV-2 and the spectrum of symptomatic SARS-CoV-2 infection ranges from mild to critical, with about 85% of patients reporting mild pneumonia and 15% with severe-to-critical illness requiring hospitalization [10]. A wide estimated range of 15–70% of patients with severe-to-critical disease will require mechanical ventilation at some point during therapy due to severe hypoxemia and ARDS [11,12].

Due to the novel nature of SARS-CoV-2, the clinical course, factors influencing progression to ARDS, predictors of outcomes, and complications both from the disease and therapy are not fully understood. Clinicians continue to rely on ongoing scientific research, anecdotal evidence, and case reports from the frontlines to guide patient care and management.

Case Reports

Patient 1

A 71-year-old man with a medical history of diabetes mellitus, hypertension, and hyperlipidemia was sent to the emergency room of an academic tertiary hospital in New York City by his primary care doctor due to concerns of COVID-19 infection. His symptoms were fever, cough, mild respiratory distress, weakness, and myalgia for about 8 days. His travel history was insignificant; however, his teenage son had recently returned from Puerto Rico and had been complaining of flu-like symptoms. His vital signs at presentation were temperature 38.5°C, pulse rate 109 bpm: blood pressure (BP) 131/73 mmHg, and oxygen saturation 92–95% on room air. He had a hemoglobin of 11.3 g/dl and white blood cell (WBC) count 10.9 cells/mm^3. His liver function tests were within normal limits. Influenza polymerase chain reaction (PCR) testing was negative. A chest X-ray (CXR) on admission showed bilateral patchy opacities concerning for pneumonia (**Figure 1A**).

Within 18 h after hospital admission, his respiratory status rapidly declined while on empiric antibiotics for atypical pneumonia and he had to be intubated and mechanically ventilated due to severe hypoxemia refractory to oxygenation by high-flow nasal cannula, prompting urgent admission to the medical intensive care unit (MICU). His nasopharyngeal swab reverse transcriptase PCR (RT-PCR) (Lenco Laboratories, Brooklyn NY) was positive for COVID-19 on day 2 of hospital admission. His CXR at this time showed worsened bilateral opacities, consistent with possible ARDS (**Figure 1B**) and PaO2/FiO2 of 156 on arterial blood gas analysis.

On day 5 of mechanical ventilation, he developed subcutaneous emphysema, pneumomediastinum, and pneumothorax (**Figure 1C**). There was no change in hemodynamic status, oxygen saturation, or peak inspiratory pressures. The patient had not undergone any thoracic procedures. No proning or recruitment maneuvers had been attempted. Lung sliding was difficult to appreciate on bedside point of care thoracic ultrasound due to the severity of the emphysema. His ventilator settings at the time were pressure control (PC) mode with FiO2 50%, PEEP 13 cmH_2O, respiratory rate (RR) 20 cpm, expired tidal volume (VTe) 527 mls (approximately 8 mls/kg), peak inspiratory pressure (PIP) 29 cmH_2O, and Pplat 20 cmH_2O. His pressure settings prior to the event are noted in **Figure 2**.

'Blow-hole' incisions were made on the skin of the anterior chest wall to decompress the severe SE, with immediate significant improvement (**Figure 1D**). The patient was extubated successfully after 2 weeks of mechanical ventilation; however, he was reintubated emergently for acute respiratory failure and died 2 days later.

Patient 2

A 58-year-old woman with a medical history of type II diabetes mellitus and hypertension was brought to the emergency room by the emergency medical services due to respiratory distress. She had complaints of one week of fever at home up to 101°F (38.3°C) and 3 days of dry cough and shortness

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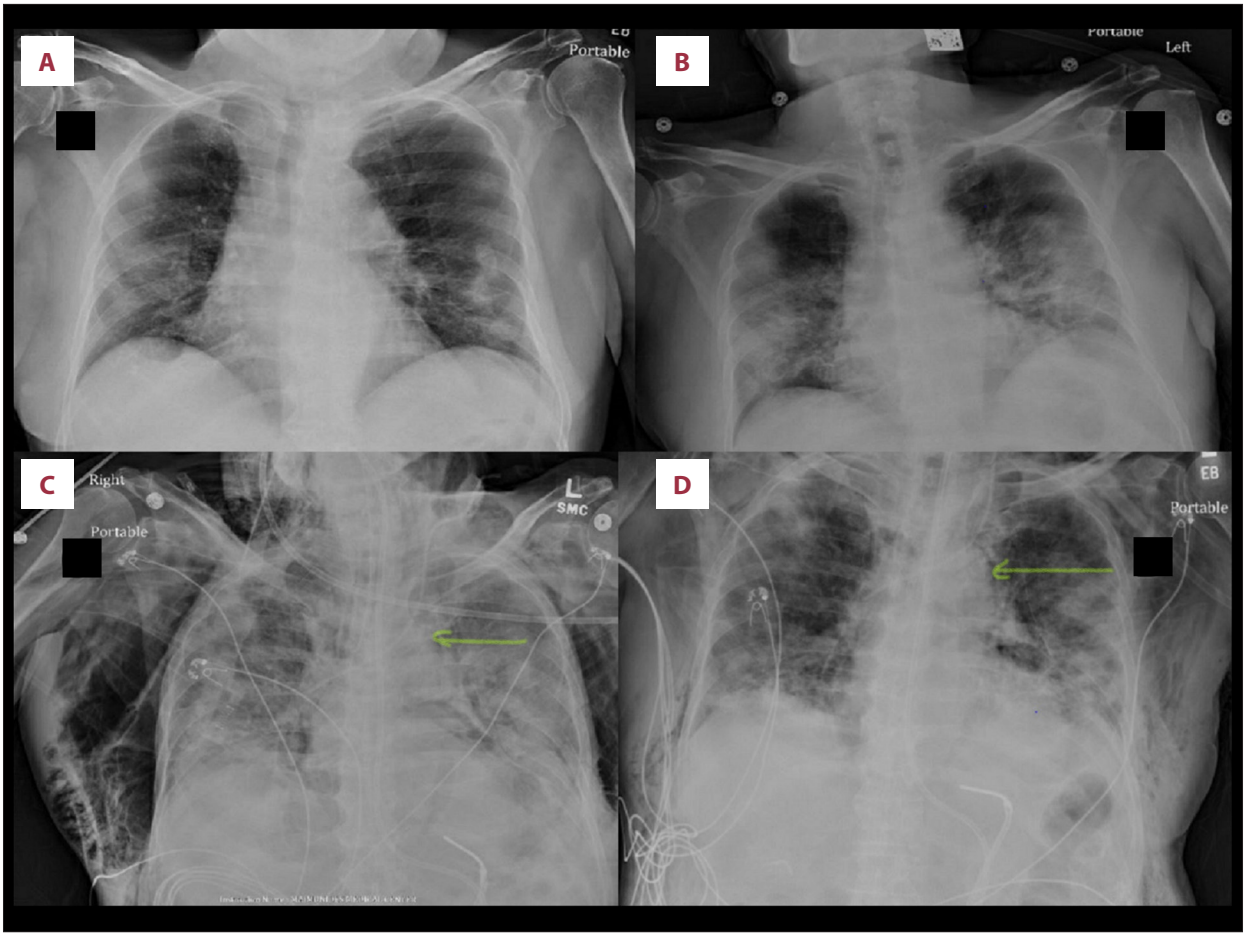

Figure 1. Patient 1 chest X-rays. (**A**) Day 1 of hospital admission – Mild bilateral lung opacities suggestive of infectious process. (**B**) Day 2 of hospital admission – Worsened bilateral lung opacities. (**C**) Day 5 of hospital admission – Subcutaneous emphysema and pneumomediastinum (yellow arrows). (**D**) Day 8 of hospital admission – Significantly improved lung opacities, subcutaneous emphysema and pneumomediastinum (yellow arrows).

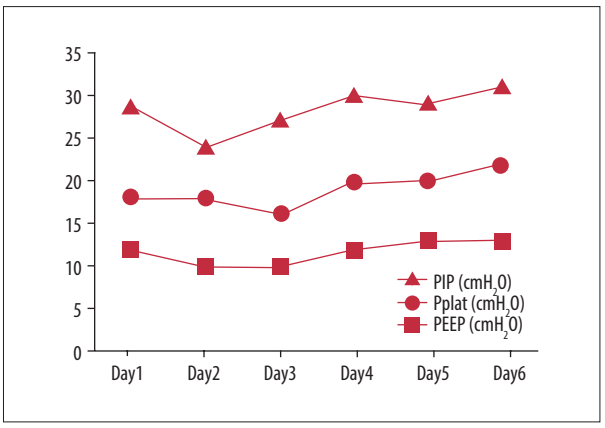

Figure 2. Patient 1 maximum ventilatory pressures from Day 1 to Day 6 on mechanical ventilation. Patient developed subcutaneous emphysema and pneumomediastinum on Day 5. PEEP – positive end-expiratory pressure; PIP – peak inspiratory pressure; Pplat – plateau pressure.

of breath. She had no recent travels or sick contacts. Vital signs on presentation were: Temperature of 102°F (38.9°C), PR: 131 bpm RR: 38 cpm, oxygen saturation of 80% on room air, weight of 55 kg and height of 150 cm. Complete blood count, basic chemistry and liver function tests were all within normal limits. Her CRP was elevated to 22.1 mg/L. Initial CXR (**Figure 3A**) demonstrated bilateral patchy opacities consistent with severe pneumonia.

She was urgently intubated and admitted to the MICU. Nasopharyngeal samples for influenza PCR test were negative while SARS-CoV-2 RNA RT-PCR by Lenco laboratories, Brooklyn NY, resulted positive. Initial ventilator settings were: Pressure-Regulated Volume Control (PRVC) mode with FiO2: 100% PEEP: 5 cmH_2O, RR: 25 cpm and TV: 500 mls. Her PaO2/FiO2 ratio was 81 in the immediate post-intubation period. Proning maneuver was commenced without any attempt at recruitment maneuvers. On the second day of mechanical ventilation, she developed extensive subcutaneous emphysema and

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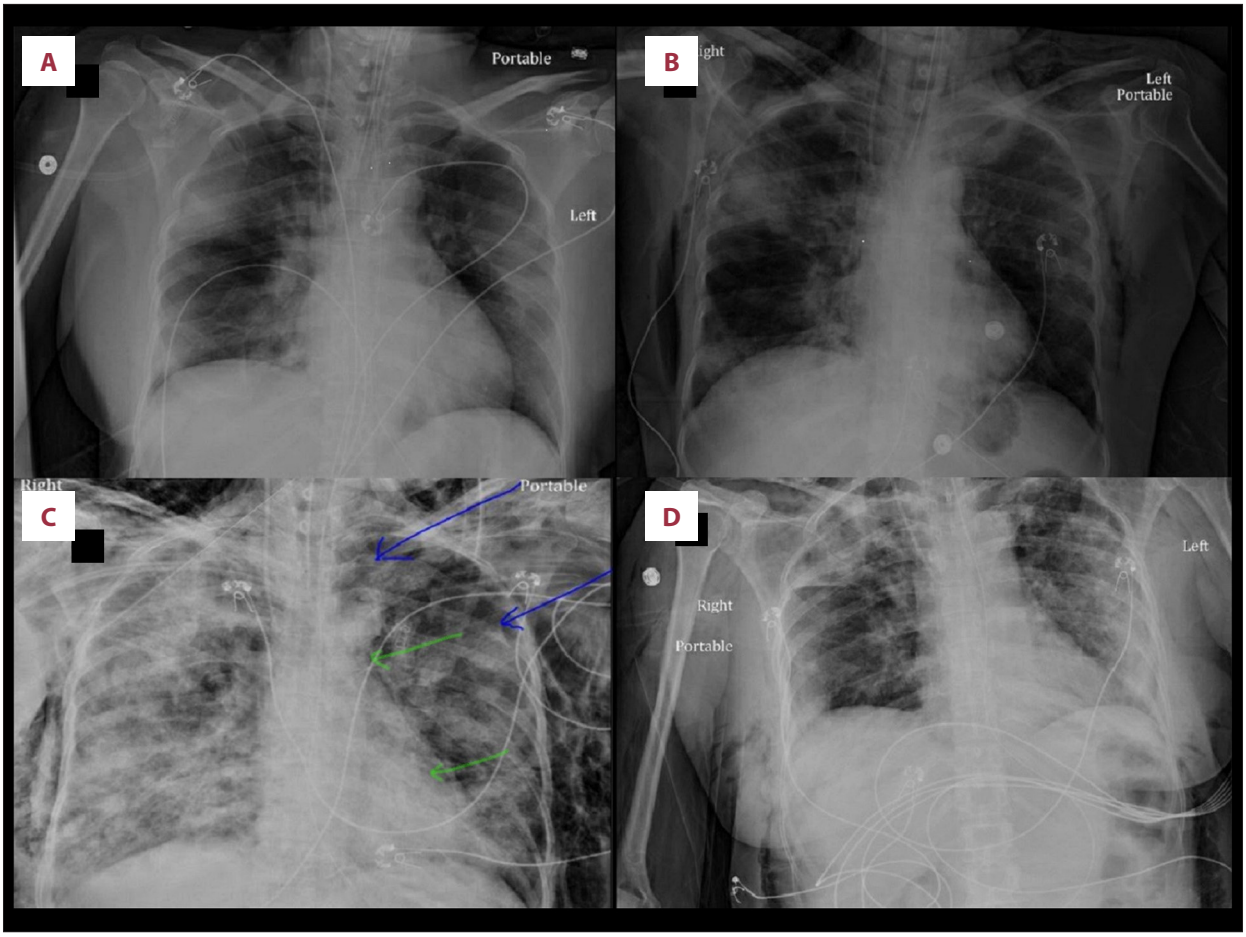

Figure 3. Patient 2 chest X-rays. (**A**) Day 1 of hospital admission – Bilateral lung opacities suggestive of severe pneumonia. (**B**) Day 2 of hospital admission – Worsened bilateral opacities with SE and pneumomediastinum. (**C**) Day 6 of hospital admission – Subcutaneous emphysema, pneumomediastinum and pneumothorax (arrows). (**D**) Day 9 of hospital admission – Significantly improved lung opacities, subcutaneous emphysema, pneumomediastinum and pneumothorax (arrows).

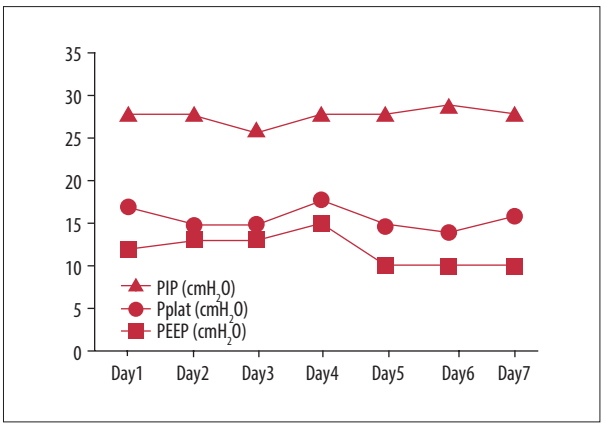

Figure 4. Patient 2 maximum ventilatory pressures from Day 1 to Day 7 on mechanical ventilation. Patient developed subcutaneous emphysema and pneumomediastinum on Day 2 and pneumothorax on day 6. PEEP – positive end-expiratory pressure; PIP – peak inspiratory pressure; Pplat – plateau pressure.

pneumomediastinum (**Figure 3B**). Her ventilator settings at the time were PEEP 13 cmH_2O and PIP 28 cmH_2O, Pplat 15 cmH_2O, and FiO2 90%, with expired tidal volume VTe: 463 mls (approximately 9 mls/kg) on PC mode.

SE was managed with 'blow-hole' incisions, with significant clinical improvement. Repeat CXR done on the sixth day of admission revealed a moderate-sized left-sided pneumothorax (**Figure 3C**). PIP at this time was 32 cmH_2O. Ventilatory pressure settings are shown in **Figure 4**. A chest tube was placed and a subsequent CXR revealed gradual resolution of the pneumothorax (**Figure 3D**). She was extubated 2 weeks after intubation and transferred to the step-down unit to recuperate.

Discussion

We have presented 2 middle-aged patients who developed barotrauma at low ventilatory pressures while receiving mechanical

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ventilation for ARDS due to COVID pneumonia. COVID-19 is an emerging and highly infectious virus responsible for the ongoing pandemic that has overwhelmed health systems globally [13]. Due to the rapid surge in infected cases requiring hospitalization, many health facilities were stretched beyond their capacities due to the overwhelming need for critical ICU resources, including personal protective equipment (PPE) for healthcare workers, ICU beds, respiratory equipment, and trained personnel. New policies regarding airway management and respiratory care techniques are particularly crucial due to high risk of infection to staff and other patients [14]. Despite the quality of care provided to these patients, morbidity and mortality rates remain very high [15,16].

In a case series of 21 patients with a median age of 72 years infected with SARS-CoV-2 in Washington state, USA, 81% were admitted to the ICU less than 24 h after admission and 72% developed severe ARDS requiring invasive mechanical ventilation [12]. The recorded mortality rate was 67% at the time of publication of the series. In another cohort of 200 patients in China, with a median age of 51 years (interquartile range, 43–60 years), 42% developed ARDS, of which 52% died [10].

A more severe course of SARS-CoV-2 complicated by moderate-to-severe ARDS has been observed in the older population [10,12,17,18]. Our patients were 71 and 58 years old and both fall within the vulnerable age group. Additionally, they both have hypertension and diabetes mellitus, which are independent risk factors for developing ARDS in SARS-CoV-2 infection [19]. The available literature suggests that the median duration from onset of symptoms and hospital admission to development of ARDS is 8 and 2 days, respectively, which is consistent with what we observed in our patients [12,18,19]. Furthermore, these patients tend to have a prolonged mechanical ventilation course, as noted in the 2 cases presented.

Our first patient developed subcutaneous emphysema with pneumomediastinum on the fifth day, while the second patient developed SE and pneumomediastinum on the second day and pneumothorax on the sixth day of mechanical ventilation. All mechanically ventilated patients are at risk of barotrauma from positive-pressure ventilation (PEEP, Pplat, and PIP) due to increased transpulmonary pressure, and the incidence rate ranges from 3% to 10% [5,20,21].

While there are conflicting data on the association between high levels of PEEP, PIP, and Pplat and barotraumas [22–24], some studies maintain that barotrauma is most likely to occur at PIP and Pplat >35 cmH_2O and static compliance <30 ml/cmH_2O [25,26]. Both of our patients developed subcutaneous emphysema and pneumomediastinum at PEEP ≤15 cmH_2O and PIP ≤35 cmH_2O, which are below the levels that have been generally reported for the occurrence of pulmonary barotrauma. Interestingly, the second patient developed pneumothorax on the sixth day of mechanical ventilation, despite maintaining relatively lower PIP and PEEP and without undergoing any predisposing procedures. A recent retrospective study showed that the incidence of barotrauma in patients with ARDS from COVID-19 was 15% compared to 0.5% in ARDS from other causes at normal ventilatory pressures [27]. It is likely that infection with SARS-CoV-2 predisposed our patients to barotrauma, despite acceptable ventilatory pressures.

We opted for the pressure control mode of ventilation while attempting to keep plateau pressure <30 cmH_2O, as recommended in ARDSNet guidelines. Pressure control mode has been found to be a convenient mode of ventilation to help reduce the work of breathing and ventilator dyssynchrony, thereby decreasing the need for neuromuscular blockade, which puts patient at more risk of critical illness myopathy [28,29]. ARDS from COVID-19 is often complicated by increased microthrombi within the alveolar circulation, leading to severe pulmonary shunting and significantly decreased gas exchange, which is compensated by high minute volumes [30]. Our patients' lungs had good compliance, which allowed for better ventilation homogeneity at acceptable ventilatory pressures. Although our patients had tidal volumes of 8–9 mls/kg, it is unlikely this led to barotrauma, because the ARDSNet study revealed no significant difference in the incidence of barotrauma between patients treated with the traditional 12 mls/kg tidal volume compared to the lung-protective strategy group treated with 6 mls/kg tidal volume [21,31].

Our patients had moderate-to-severe ARDS, which is an independent predictor of barotraumas [5,21]. They did not have any other known or documented risk factors, including underlying chronic lung diseases such as COPD, asthma, ILD, or significant tobacco smoking. Both patients did not suffer any significant mucosa injuries during trachea suction. We believe that SARS-CoV-2-associated ARDS predisposed our patients to barotrauma, possibly through coronavirus-induced alveolar damage. Zhang et al described spontaneous SE and pneumomediastinum in a patient with H5N6 (avian) flu at a PEEP of 15 cmH_2O and PIP of 40 cmH_2O [32]. Also, Luis et al. [33] and Padhy et al. [34] reported cases of spontaneous pneumomediastinum and subcutaneous emphysema not related to mechanical ventilation in patients infected with H1N1 (swine) flu. Again, it is quite possible that there is a relationship between severe respiratory viral infections and development of barotraumas, and this could be a subject of further research.

Patients with moderate-to-severe ARDS often require a high PEEP to overcome refractory hypoxemia and to prevent atelectrauma from the shear forces caused by the repetitive opening and closing of the alveoli [35]. In a study of mechanically ventilated patients with SARS-Cov-2 admitted to an ICU in Italy,

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the median PEEP required was 14 cmH_2O. Recent guidelines published by the 'Surviving Sepsis Campaign' on the management of critically ill adults with COVID-19 also recommended using a higher PEEP strategy and discouraged incremental PEEP recruitment maneuvers [36]. The benefits of high PEEP in moderate-to-severe ARDS appear to outweigh the risk of barotrauma, and we suggest that patients should be treated with appropriate PEEP to prevent de-recruitment of alveoli and to improve outcomes.

Pulmonary barotrauma can complicate mechanical ventilation in the management of patients with ARDS. As we continue to learn from the evolving clinical situation of COVID-19 and associated moderate-to-severe ARDS, clinicians should be aware of pulmonary barotrauma as a possible complication, even at low ventilatory pressures. Pulmonary barotrauma is generally prevented by low tidal volume ventilation, appropriate PEEP application, and maintaining low Peak and Pplat pressures according to standard guidelines. Close monitoring for barotrauma and prompt recognition has been shown to improve outcomes.

Conclusions

It is quite plausible that infection with SARS-CoV-2 makes patients more susceptible to mechanical ventilation-induced barotrauma, even at low ventilatory pressures.

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Conflicts of interest

None.

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