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CASO CLÍNICO

Barotrauma: An Additional and Relevant Concern in COVID-19 Patients

Barotrauma: Uma Preocupação Adicional e Relevante nos Doentes com COVID-19

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Afiliação
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ABSTRACT

Invasive mechanical ventilation plays a key role in the management of respiratory failure induced by SARS-CoV-2 infection. Patients with COVID-19 have been associated with need for both long periods of ventilation and higher pressures applied to a often damaged lung parenchyma. These features may prompt an increased risk of lung injury, namely barotrauma, even when a lung protective ventilation strategy is applied. We report a case of a COVID-19 ventilated patient who developed several barotrauma related complications that ultimately led to death.

RESUMO

A ventilação mecânica invasiva tem um papel fundamental na abordagem da insuficiência respiratória induzida pela infeção por SARS-CoV-2. Os doentes com COVID-19 têm demonstrado necessidade de longos períodos de ventilação e de pressões elevadas, muitas vezes aplicadas a um parênquima pulmonar lesado em contexto da própria infeção. Estas características podem promover um risco acrescido de lesão pulmonar, nomeadamente barotrauma, mesmo quando é aplicada uma estratégia ventilatória pulmonar protetora. Apresentamos o caso de um doente ventilado com COVID-19 que desenvolveu várias complicações relacionadas com o barotrauma, as quais culminaram no seu falecimento.

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INTRODUCTION

The overdistension and high pressures generated during invasive mechanical ventilation (IMV) can cause bronchial or alveolar rupture leading to barotrauma. The consequent air leak can occur into the mediastinum (pneumomediastinum), pleural cavity (pneumothorax), subcutaneous tissues (subcutaneous emphysema) and peritoneal cavity (pneumoperitoneum). The risk of barotrauma is increased in patients with severe underlying lung disease, namely acute respiratory distress syndrome (ARDS).[1] Since the ARDSnet trial, lung-protective ventilation strategies (tidal volume 6 mL/kg of predicted body weight and plateau pressure < 30 cmH_20) have been implemented to reduce complications of IMV in patients with acute lung injury and ARDS.[2] Barotrauma is associated with a significant increase in the intensive care unit (ICU) lenght of stay and mortality.[3] Recently, it was reported a prevalence of barotrauma of 40% among coronavirus disease 2019 (COVID-19) patients under IMV and it was found that in these patients barotrauma is an independent risk factor for death and is associated with a longer hospital lenght of stay.[4,5] Nevertheless, it has also been described an increased risk of pneumothorax, pneumomediastinum and subcutaneous emphysema even in unventilated COVID-19 patients.[6-9] In these patients it has been proposed that strutural changes that occur in the lung parenchyma caused by SARS-CoV-2 infection – namely cystic and fibrotic changes – may lead to bulla formation, wich in turn when associated to the increased intrathoracic pressure resulting from prolonged cough,, may cause pneumothorax and potentially pneumomediastinum and subcutaneous emphysema.[8,9] We report a case of a COVID-19 patient who received IMV and developed several barotrauma related complications that ultimately may have contributed to death.

CASE REPORT

A 62-year-old male with arterial hypertension treated with irbesartan and no history of smoking or previous respiratory disease presented to the Emergency Department with dry cough and fever for one week. On admission, he had mild hypoxemia (PaO_2/FiO_2 257 mmHg), lymphopenia (630/µL) and a C-reactive protein of 4.3 mg/dL. Chest X-ray revealed bilateral interstitial pulmonary infiltrates. Nasopharyngeal swab tested positive for SARS-CoV-2 in a real-time polymerase chain reaction assay. The diagnosis of pneumonia induced by SARS-CoV-2 was made and the patient was admitted to a general coronavirus ward and started oxygen therapy and treatment with hydroxychloroquine and lopinavir/ritonavir.

On the fifth day (D5), the patient evolved into septic shock and hypoxemic worsening (PaO_2/FiO_2 182 mmHg) requiring ICU admission, where he was intubated and started on IMV in a volume control mode with a lung-protective strategy (6-8 mL/kg predicted body weight). A good lung dymamic compliance (55-90 mL/cmH_20), plateau preassure < 30 cmH20 and driving pressure < 15 cmH_20 were mantained as long as the patient was under IMV. On the fifteenth day (D15), hematic drainage through the nasogastric tube was noted. Upper gastrointestinal endoscopy (UGE) revealed erythematous gastropathy without active bleeding. Shortly after the UGE, the patient developed subcutaneous emphysema without impairment in gas exchanges. Chest computed tomography (CT) with oral contrast revealed cervical subcutaneous emphysema, large pneumomediastinum and small pneumoperitoneu (Fig. 1) without pneumothorax or esophageal leakage of oral contrast.

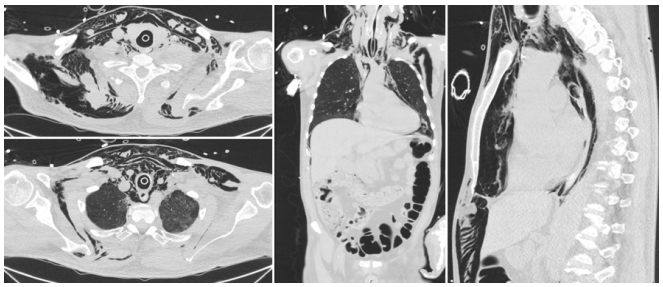

Figure 1. CT scan images revealing cervical subcutaneous emphysema dissecting several muscle groups and communicating with large pneumomediastinum. Sagittal view shows small pneumoperitoneum in the anterior abdominal plans

The patient underwent flexible bronchoscopy which ruled out tracheoesophageal fistula and repeated UGE wich definitely excluded esophageal perforation or other iatrogenic lesions. A nonsurgically approach was decided (oral intake cessation, nasogastric tube draining and parenteral nutrition) and micafungin and broad-spectrum antibiotics (meropenem and vancomycin) were started to prevent a mediastinitis. On the twentieth day (D20), radiological reassessment showed significant improvement both in subcutaneous emphysema and pneumomediastinum and feeding through nasogastric tube was re-introduced without complications.

For the next two weeks the patient remained in the ICU and it was possible to stop the vasopressor support and progress in sedation weaning. However, on the thirty-six day (D36) after hospital admission, he started fever again and increasing blood inflammatory markers (C-reactive protein 14.90 mg/dL and procalcitonin 16.09 ng/mL). Upon suspicion of infection, right jugular central venous catheter was removed and an ultrasound-guided central venous catheter was placed in the left jugular vein. Less than an hour later, the patient developed progressive desaturation. Chest X-ray revealed right-sided pneumothorax (Fig. 2) and a chest tube was placed allowing air drainage from pleural space.

Chest CT additionally revealed worsening ground glass opacification and previously absent consolidation areas.

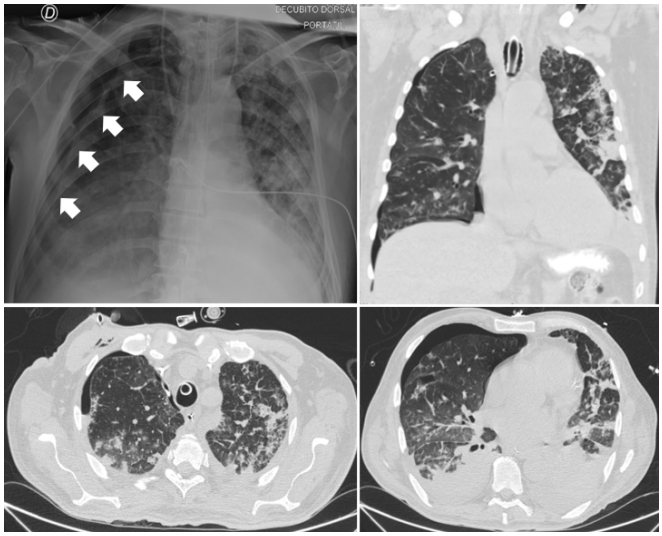

Figure 2. In the upper left corner, chest X-ray showing right-sided pneumothorax (arrows). CT performed after chest tube placement revealing right pneumothorax, bilateral pleural effusion, worsened ground glass opacities and crazy-paving pattern with previously absent consolidation areas

Based on fever onset, increasing inflammatory markers and new pulmonary infiltrates, it was assumed a nosocomial respiratory infection, namely a ventilator-associated pneumonia. New blood cultures and tracheal aspirate were collected for microbiological analyze and a new cycle of antibiotics with tigecycline and vancomycin was empirically started. Unfortunately, the patient deteriorated with multiorganic dysnfunction (respiratory, cardiovascular and renal) with increasing need for vasopressor and ventilatory support and died a few hours later. One weak after death, the result of blood cultures was released revealing the isolation of Pseudomonas aeruginosa in all samples collected. We assumed that the patient died because of refractory septic shock caused by ventilator-associated pneumonia and Pseudomonas aeruginosa bloodstream infection. Ultimately,

pneumothorax contributed to this unfavorable outcome because it was an additional cause of respiratory dysfunction.

DISCUSSION

Barotrauma is a recognized complication of mechanical ventilation.[10] In this case report, the patient developed barotrauma in two different moments - first with pneumomediastinum, subcutaneous emphysema and pneumoperitoneum after UGE performed for suspected gastrointestinal bleeding and later with pneumothorax after jugular central line placement. In both moments these events most likely occured as complications of mechanical ventilation since in the first event tracheoesophageal fistula or other iatrogenic lesions were ruled out by bronchoscopy and repeated UGE and in the second case pneumothorax developed on the contralateral side of the jugular catheter placement. Assuming that these events were actually barotrauma complications, we may question its occurence since a lung protective strategy was employed from the beggining. This may rise the question if SARS-CoV-2 infection poses a particularly high risk of barotrauma. Diffuse alveolar damage has been described as a key pathophysiologic mechanism in new coronavirus induced lung injury.[11] Some authors have suggested that alveolar damage caused by this viral infection leads to severe destruction of alveolar tissue, resulting in bulla formation in formerly healthy lungs and enhancing the risk of pneumothorax and other barotrauma presentations. This hypothesis is supported by the fact that an increased incidence of pneumothorax has been reported in both ventilated and unventilated COVID-19 patients.[8,9]

Additionally, COVID-19 patients demand long periods of ventilation with more time of exposure to airway positive pressure and they may be difficult to ventilate as they often present decreased respiratory system compliance mimicking ARDS and lung fibrosis.[10,12] Lung perfusion regulation compromisse and thrombotic microvascular injury may further contribute to the pathophysiologic mechanisms in COVID-19 and might lead to severe hypoxemia in patients with preserved lung complicance.[13,14] Mechanical ventilation itself can further aggravate or even induce lung injury highlighting the role of ventilation strategies to protect from excessive lung stress and strain.[10] For all these reasons COVID-19 patients may be at particularly increased risk of barotrauma even under protective lung ventilation and regardless of previous history of smoking or respiratory disease. In a recent retrospective case-control study, barotrauma in COVID-9 induced respiratory failure requiring mechanical ventilation was found in 40% of the patients. Patients who developed barotrauma (8/20) had a median age of 62 years and a previous median time of IMV of 18 days before barotrauma. Complications included pneumothorax (n=5), pneumomediastinum (n=5), extended subcutaneous emphysema (n=2) and pneumopericard (n=1), with four patiens showing more than one type of complication. Only one patient presented predisposing lung disease (chronic obstructive lung disease). Interestingly, the authors reported lower pressures and tidal volumes in patients with barotrauma compared to patients without barotrauma, reaching statistical significance for the comparison of peak pressure and tidal volume, suggesting no direct association between the occurence of barotrauma and the applied ventilatory parameters when following recommendations for lung protective ventilation.[4] Analog to the patients in that study, the patient we presented had no history of respiratory disease and although he was ventilated according to lung protective recommendations he still developed several barotrauma complications. Previously, Anzueto A *et al* had also found no correlation between barotrauma and mechanical ventilation parameters.[3] In many parts of the world, COVID-19 pandemic required many health professionals without training in mechanical ventilation to be involved in the treatment of these patients in intermediate and intensive care units. This clinical case also intends to highlight the role of safely programming the ventilator settings according to a protective lung strategy in order to no further increase the risk of barotrauma. Another strategy to minimize the risk of barotrauma is early ventilatory weaning, protecting lung for prolonged positive pressure ventilation.

In conclusion, barotrauma is a complication of mechanical ventilation of particular concern in COVID-19 ventilated patients even when recommendations for lung protective ventilation are implemented. Lung damage elicited by SARS-CoV-2 infection, prolonged need for mechanical ventilation and ventilator-induced lung injury may increase the risk and amplify the extention of barotrauma. Healthcare providers must be able to early recognize this complication and manage it properly.

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