Journal of Community Hospital Internal Medicine Perspectives

Volume 12 | Issue 3 Article 11

2022

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Recommended Citation

Mohammadian, Mahsa; Mowafy, Ahmed; and Dikhtyar, Anthony (2022) "Successful Utilization of Life-saving Extra-Corporeal CO2 Removal in Catastrophic Status Asthmaticus: a Case Report," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 12: Iss. 3, Article 11.
DOI: 10.55729/2000-9666.1056
Available at: https://scholarlycommons.gbmc.org/jchimp/vol12/iss3/11

CASE REPORT

Successful Utilization of Life-saving Extra-Corporeal CO2 Removal in Catastrophic Status Asthmaticus: A Case Report

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Abstract

Acute asthma exacerbations can be severe and life-threatening. In some cases, standard interventions and management do not result in reversal of bronchoconstriction. It is crucial to detect patients with impending respiratory failure and escalate management to invasive mechanical ventilatory support and, in refractory cases, interventions like extracorporeal membrane oxygenation (ECMO). This technique is not frequently utilized but has proven to be effective in settings of resistant status asthmaticus.

We describe a Case of respiratory distress secondary to asthma exacerbation, which rapidly devolved into status asthmaticus. It was resistant to all standard and off-label management modalities, which necessitated the use of veno-venous extracorporeal CO2 removal (VVECCO2R). ECMO was utilized in our case with great success. In this article, we aim to raise awareness of the importance of VVECCO2R in the treatment of refractory status asthmaticus and the difficulties that prevent widespread implementation of the technique across healthcare facilities.

Keywords: Status asthmaticus, Extra-corporeal CO2 removal, Refractory asthma

1. Introduction

Asthma is managed with short-acting beta-receptor agonists, long-acting beta-receptor agonists, and corticosteroids as mainstay therapy. However, acute asthma exacerbations can become refractory to those therapies, necessitating the use of invasive mechanical ventilation to provide the patient with adequate airflow. Approximately 4% of patients hospitalized with asthma exacerbations require this invasive intervention.[1]

Status asthmaticus is a severe form of asthma exacerbation that does not respond to the usual treatment modalities of reactive bronchoconstriction. It can last from minutes to several hours and can be a life-threatening condition even when properly managed. Risk factors for status asthmaticus include previous ICU admission and recent increased use of bronchodilator treatment without symptom improvement. In patients with severe asthma refractory to standard medical treatment and mechanical ventilation, extracorporeal life support can provide a means of carbon dioxide removal and oxygenation.

We present a Case of acute severe asthma exacerbation that failed to resolve despite the use of all recommended and off-label lines of treatment, requiring the utilization of extracorporeal gas exchange.

2. Case presentation

A 17-year-old male with a past medical history of asthma and psoriasis presented to the emergency department (ED) with complaints of progressive shortness of breath over the preceding 3 days. The patient had a long history of asthma since childhood, with multiple emergency room visits. The patient was under close follow with a pulmonologist due to uncontrolled asthma. He was previously educated on inhaler use and the importance of

Received 11 July 2021; revised 13 December 2021; accepted 16 December 2021.
Available online 2 May 2022

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https://doi.org/10.55729/2000-9666.1056

remaining compliant with his medication. The family reported that he was occasionally not compliant with the inhalers. His home medication was adjusted multiple times and included umeclidinium/vilanterol 62.5–25 mcg/inh inhalation powder daily, cetirizine 10 mg daily, prednisone 40 mg oral daily, and albuterol sulfate as needed. The patient reported using the rescue inhaler once daily for the preceding three weeks. However, for two days prior to his presentation in the ED, he admitted to using the inhaler every 2–3 h with no improvement. At the time of presentation to the emergency room, he was in respiratory distress, vomiting, cyanotic, and unable to speak in full sentences. Physical examination revealed severe bilateral biphasic wheezes.

The patient was treated with 125 mg of methylprednisolone and 2 gm of IV magnesium sulfate, before being intubated in the emergency department secondary to severe respiratory distress, impending respiratory failure, and intractable vomiting. The patient was admitted to the ICU, ventilated under pressure regulated volume control, PEEP of 5, FiO2 100%, tidal volume 400, respiratory rate 20, O2 saturation 100%. Chest x-ray obtained post-intubation revealed an endotracheal tube in place, with no acute pulmonary findings [Fig. 1].

The ABG obtained post-intubation revealed a pH of 7.09 (7.35–7.45), PaO2 337 mmHg (>80 mmHg), PaCO2 91 mmHg (35–45 mmHg), HCO3 21.3 mmol/L (22–26 mmol/L). Initial labs revealed a Hgb 15 gm/dL (12–16 gm/dL), WBC 24.7 (4.8–10.8), with absolute neutrophil count of 9.3 (1.4–6.5), eosinophils 1.4 (0.0–0.7), and lymphocytes 1.4 (1.2–3.4).

The patient's peak airway pressure was increasing, reaching as high as 50 mmHg, so his ventilation was switched to pressure-control to prevent pulmonary barotrauma. At that time the patient was sedated and was started on a vecuronium infusion. Albuterol-Ipratropium-Inhalation dose was increased to 3 mL every 2 h via nebulizer before the initiation of the vecuronium. The patient was also treated with epinephrine 0.5 mg intramuscular and terbutaline 0.25 mg subcutaneous for 3 times in an effort to decrease his airway resistance.

A second ABG revealed worsening of the condition, with a pH of 6.91, PaO2 124, PaCO2 146, and HCO3 19.2. The patient was given bicarbonate pushes and started on a bicarbonate infusion for severe acidosis. A repeat ABG 2 h later showed no significant improvement, with a pH of 6.97, PaCO2 141 and HCO3 of 22. We then added theophylline to the therapy, which failed to improve the patient's airway resistance. The ABG 1.5 h after the initiation of theophylline, showed worsening of the hypercapnia, with a pH of 6.94, PaCO2 156 and incalculable HCO3.

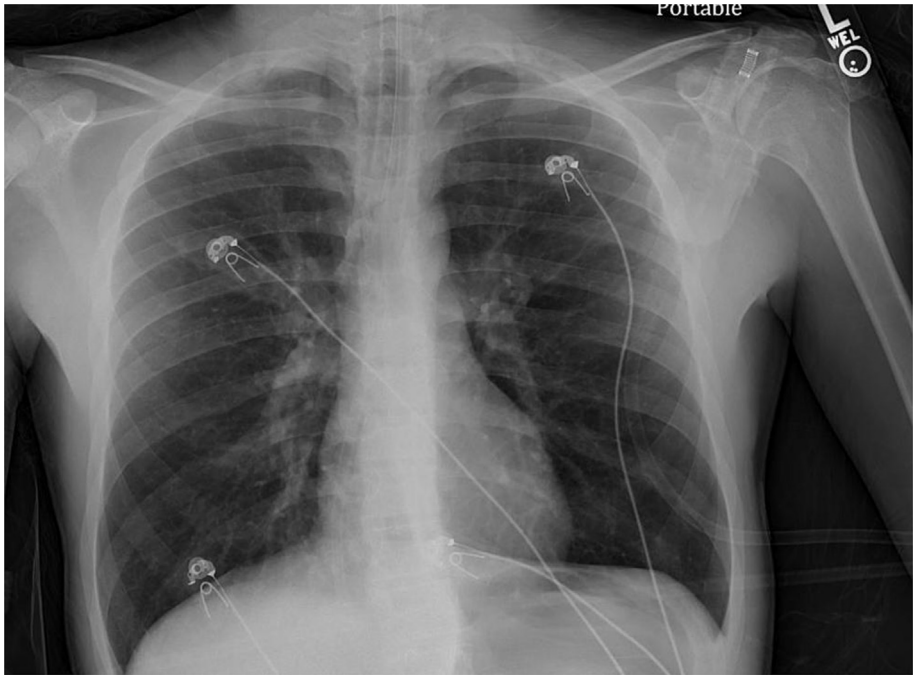

Fig. 1. Chest x-ray demonstrating hyperinflated lungs despite intubation.

With pressure-control ventilation, the tidal volume dropped to around 150 ml, and minute ventilation dropped to 3000 ml/min. The following ABG 2 h later, revealed a pH 6.93, PaO2 210, PaCO2 199, and incalculable HCO3, while O2 saturation remained at 100%. The patient was given a ketamine push, 0.5 mg/kg, followed by a ketamine infusion, 0.5 mg/kg/hr, which also failed to decrease airway resistance. PaCO2 remained at 198 after 1 h.

A follow-up chest x-ray showed signs suggestive of a pneumomediastinum, which was expected given the patient's exceedingly high airway pressures [Fig. 2].

An electrocardiogram was obtained, which demonstrated low voltage readings, agreeable with the state of lung hyperinflation [Fig. 3].

The patient was too unstable to transfer to the operating room to be put under general anesthesia with inhaled sevoflurane. At that time, the patient was only eligible for veno-venous extracorporeal carbon dioxide removal (VVECCO2R).

Our facility is not readily equipped to deploy VVECCO2R, so several communications were made in an attempt to transfer the patient to another facility where the technique is more standardized. He was accepted to a facility but was too unstable to transport. A specialized team from the accepting facility came to our facility's Intensive Care Unit and prepared the patient for extracorporeal gas exchange. The patient was started on VVECCO2R at the bedside. An ABG obtained 30 min later revealed an improved pH of 7.12, PaO2 125, PaCO2 120. Another ABG at 60 min after VVECCO2R revealed a pH of 7.25, PaO2 120, PaCO2 89. Total duration of hospitalization in our facility was 20 h before we transferred the patient to a hospital that is better equipped to manage a patient on VVECCO2R.

He remained on VVECCO2R for 21 days. He was then transitioned back to mechanical ventilation for another 30 days. The patient was successfully weaned off invasive ventilation and discharged from the hospital to respiratory rehabilitation. A follow-up chest x-ray demonstrated atelectasis and possible consolidation after prolonged invasive mechanical ventilation [Fig. 4].

3. Discussion

Traditionally, asthma exacerbations that are resistant to pharmacological interventions are managed with non-invasive and invasive ventilator support. Optimizing ventilator settings to maximize CO2 removal and decrease hyperinflation can be challenging as several complications can occur. These include sepsis, ARDS, elevated intrathoracic pressure, right ventricular strain causing inadequate preload, and positive pressure ventilation leading to tension pneumothorax.[2] Additionally, airway stenosis and subsequent air trapping in severe asthma

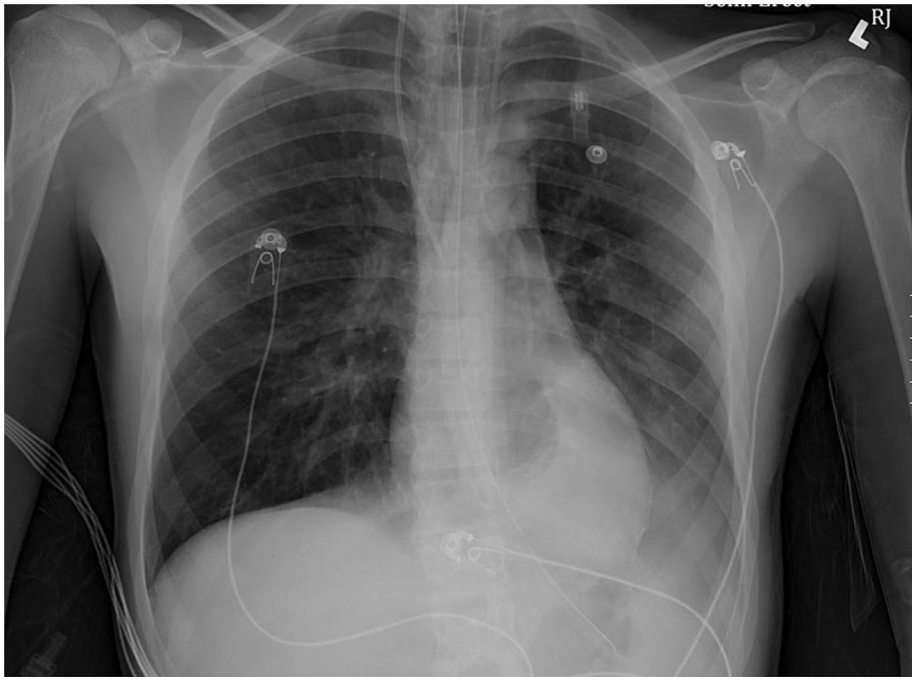

Fig. 2. Chest x-ray demonstrating worsening of pulmonary inflation despite mechanical ventilation and treatment to bronchoconstriction, with possible pneumomediastinum.

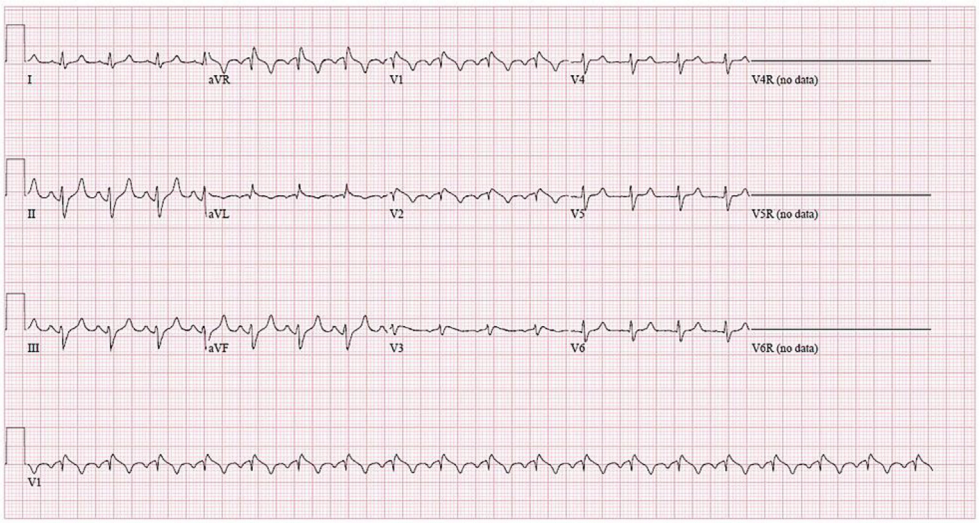

Fig. 3. A low voltage EKG obtained during management, demonstrating a hyperinflated chest.

can limit the effectiveness of mechanical ventilation altogether. In these scenarios, extracorporeal membrane oxygenation (ECMO) may be utilized to provide gas exchange and adequate carbon dioxide removal with minimal to no reliance on a ventilator.[3]

ECMO is a method of providing tissue oxygenation and perfusion in settings of inadequate respiratory or cardiovascular function. Veno-arterial ECMO (VA-ECMO) bypasses both the heart and lungs and is used in settings where simultaneous hemodynamic and respiratory support is needed. Veno-venous ECMO (VV-ECMO) is used in settings of preserved cardiac function as it offers only respiratory support. VV-ECMO has historically been used to manage acute respiratory distress syndrome (ARDS), but since 1992, it has gained popularity as a treatment for severe asthma exacerbations, perhaps because of its favorable complication profile.[4] Even so, hemorrhagic, renal, and cardiovascular

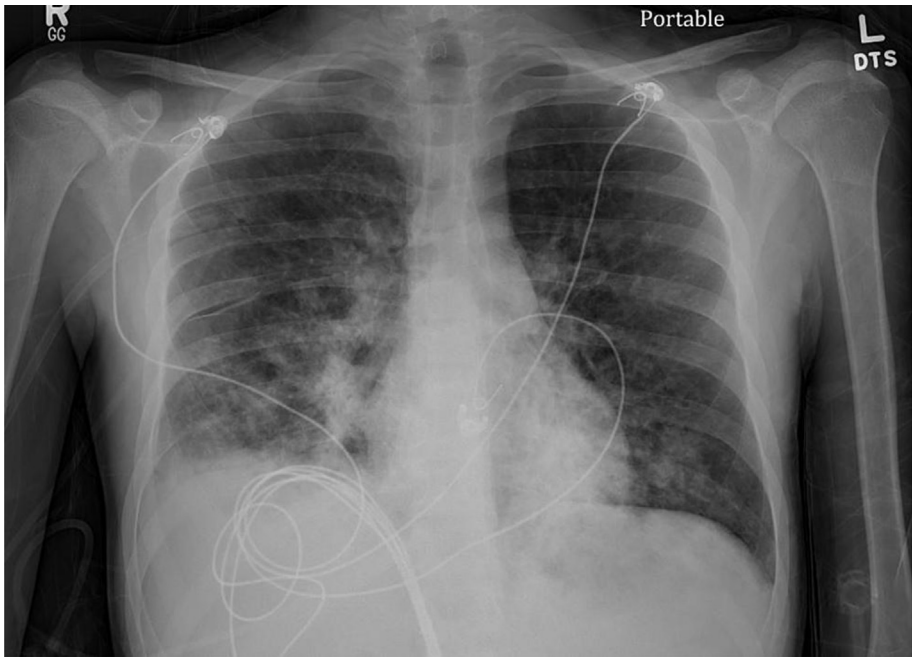

Fig. 4. A chest x-ray obtained after the patient's recovery, demonstrating atelectasis and possible consolidation after prolonged invasive mechanical ventilation.

complications have been noted with its use.[5] Current indications for ECMO include hypoxemic respiratory failure with PaO_2/FiO_2 <100 mmHg despite optimized ventilator settings, and hypercapnic respiratory failure with an arterial pH < 7.20.[6,7]

In the literature, a survival rate of 83.5% has been reported for patients with severe asthma exacerbations undergoing VV-ECMO. Compared to survivors, non-survivors had significant differences in age, pH, PEEP, ECMO duration, and complication rate. Importantly, respiration rate, fraction of inspired oxygen, peak inspiratory pressure and mean airway pressure significantly improved in patients who underwent ECMO after mechanical ventilation failed.[5] Unfortunately, ECMO is not widely used to treat status asthmaticus because of multiple obstacles that include lack of equipment at healthcare facilities, ill-equipped staff, and transportation limitations. Given the benefits and favorable complication profile, ECMO should be more accessible and widely used to manage status asthmaticus refractory to ventilator support.

4. Conclusions

ECMO remains a lifesaving option in cases of status asthmaticus with severe hypercapnic respiratory failure unresponsive to medical treatments and mechanical ventilatory support. Increasing physician awareness of ECMO indications, equipping hospitals with proper equipment, and training healthcare staff could make this modality more ubiquitous and improve patient outcomes.

Funding

The authors have no funding to disclose.

Conflict of interest

The authors declare no potential conflicts of interest.

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