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Case Review

A 63-Year-Old Male Interfacility Transfer for Extracorporeal Membrane Oxygenation Evaluation

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A 63-year-old white male patient with a history of hypertension and hyperlipidemia and a remote history of cervical neck cancer and tuberculosis presented to an urgent care clinic with a complaint of dyspnea with an associated cough productive of rust-colored sputum, fevers, and myalgia for several days. Despite over-the-counter treatments, the patient reported that his cough was worse at night and was increasing in severity. He continued to be compliant with his home medications, which included hydrochlorothiazide (25 mg), lisinopril (10 mg), and pravastatin (40 mg). In the urgent care clinic, his examination was significant for the following vital signs: blood pressure of 128/74, heart rate of 108, respiratory rate of 18, SpO_2 of 95% on room air, and an oral temperature of 99.8°F. Rapid influenza A antigen and B antigen tests were negative. He was diagnosed with bronchitis and prescribed albuterol (90 μg), benzoate (100 mg), guaifenesin (600 mg), and ibuprofen (400 mg).

Four days later, his family noticed that he had difficulty breathing with hemoptysis. Additionally, he was lethargic, responded sluggishly to verbal stimuli, and was confused. Emergency medical services were called, and on the initial assessment, he was noted to be hypoxic with an oxygen saturation of 76% despite being placed on a nonrebreather mask at 15 L/min. He was also noted to be hypoglycemic with a capillary blood sugar of 50 mg/dL. Intravenous access was obtained, and the patient was given an amp of dextrose 50 (25 g) with no improvement in mentation. He was transported to a regional medical center for further evaluation and treatment.

In the emergency department (ED), the patient was noted to have a Glasgow Coma Scale of 11 (E-2, V-4, M-5) with a repeat blood sugar of 64 mg/dL and a significantly elevated respiratory rate at 30 breaths per minute; increased work of breathing hindered the patient from speaking more than 1 to 2 words at a time with significant hypoxia (peripheral O_2 saturation of 64% on high-flow oxygen). An additional amp of dextrose 50 was administered, and bilevel positive airway pressure was initiated at 14/8 and 100% fraction of inspired oxygen (FiO_2) with no improvement. Laboratory values and cultures were drawn (Table 1).

On physical examination, the ED staff noted that the patient was pale with delayed capillary refill. Lung sounds were significant for diffuse coarse rhonchi throughout all fields. Additionally, the patient's mentation continued to wane. He was subsequently orally intubated because of failure to oxygenate; 20 mg etomidate was given intravenously, and laryngoscopy showed a grade I view. The endotracheal tube (ETT) was inserted on the first attempt and secured at 21 cm at the corner of the mouth. No paralytics were given. Postintubation, respiratory staff suctioned copious amounts of "cola-colored" secretions from the ETT, and the position was again confirmed via auscultation. End-tidal carbon dioxide waveform capnography and revisualization with a video laryngoscope confirmed the ETT to be in a good position. He was then placed on a ventilator on assist control with a respiratory rate of 18, a tidal volume of 550, FiO_2 of 100%, and a positive end-expiratory pressure of 5. A total of 1 L cola-colored secretions was suctioned from the ETT. The initial chest x-ray noted bilateral opacities with no present pneumothorax or hemopneumothorax confirming the impression of bilateral pneumonia.

Table 1
Laboratory Values of a 63-Year-Old Man in Respiratory Distress

CBC		CMP	
WBC	0.3	Na	134
Hgb	13.3	K	4.9
Hct	41	BUN	74
Platelets	162	Cr	3.51
		Cl	102
		CO_2	20
Lactate	4.8	Glucose	255
pH	7.1		

BUN = blood urea nitrogen; CBC = complete blood count; Cl = chloride; CMP = comprehensive metabolic profile; Cr = creatinine; Hgb = hemoglobin; Hct = hematocrit; WBC = white blood cell.

Additional peripheral intravenous lines (×4) were secured, and a 7F left internal jugular triple-lumen central venous catheter was inserted. Fluid resuscitation was initiated with a total of 3 L 0.9% normal saline and 2 L lactated Ringer solution. Broad-spectrum antibiotics were administered including 3.375 g piperacillin/tazobactam, 1.0 g vancomycin, and 900 mg clindamycin. Because of the patient's hemodynamic instability, only intermittent doses of lorazepam were given for sedation. A fentanyl drip was initiated for analgesia.

At this point, the patient continued to have worsening hemodynamic instability. The patient's heart rhythm had converted from sinus tachycardia with heart rates in the low 100s to atrial fibrillation with variability into the 110s to 120s. Unfortunately, he additionally had bursts of supraventricular tachycardia in the 150s. In order to combat the worsening cardiac irritability, the sending facility initiated a 150-mg bolus of amiodarone over the course of 10 minutes. It resulted in

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Table 2
The Initial Arterial Blood Gas (ABG) of a 63-Year-Old Man in Multisystem Organ Dysfunction

ABG Values	
pH	7.10
$PaCO_2$	62
PaO_2	58
FiO_2	1.0
HCO_3	19.0
BE	−11.4

BE = base excess; FiO_2 = fraction of inspired oxygen; HCO_3 = bicarbonate.

appropriate rate control but also led to significant hypotension unresponsive to fluid boluses.

With a blood pressure of 57/42 despite 3 L crystalloid, norepinephrine, vasopressin, and neosynephrine were initiated sequentially to sustain hemodynamic stability with little success. An arterial blood gas test was completed at this point and is summarized in Table 2.

In addition, the sending facility initiated steroids (250 mg methylprednisolone intravenously), administered a sodium bicarbonate infusion for acidosis, and infused 2 units of packed red blood cells for the treatment of anemia in the setting of marked hypotension. Because of the grave condition of the patient, a regional tertiary care medical facility was contacted for referral for extracorporeal membrane oxygenation (ECMO) evaluation, and the health system's flight team was subsequently alerted to the mission.

Upon arrival, the flight team found the patient in an ED examination room crowded with staff. The flight team performed an initial examination and noted the ETT to be in a good position confirmed via multiple methods including a chest x-ray, end-tidal carbon dioxide monitoring, and physical examination. They suctioned an additional 200 mL cola-colored secretions from the ETT and noted that the patient remained significantly hypotensive and hypoxic on the current ventilation settings. The referring facility's infusions were transferred to the flight team's pumps. Vasopressors and fluids were titrated, and a third unit of packed red blood cells was initiated per the receiving pulmonologist. Additionally, the receiving critical care team requested that the flight team continue with blood administration (2 units), chemical paralysis for the duration of the flight for ease of ventilation and oxygenation, and the administration of nebulized epoprostenol for improved oxygenation during the course of the flight as a bridge therapy for ECMO.

During the course of the flight, an additional 300 mL cola-colored secretions was suctioned from the ETT. The flight to the receiving facility was tasked with the team addressing profound hypotension and hypoxia with titrations in vasoactive medications, fluid administration, and ventilator adjustments in an attempt to optimize oxygenation while preserving hemodynamics.

The patient was delivered to the tertiary care facility remaining in extremis. Although the patient was admitted to the heart and vascular intensive care unit, a bedside bronchoscopy was performed to rule out the possibility of a bronchoesophageal fistula. The patient was diagnosed with septic shock and multiple organ dysfunction syndrome secondary to hemorrhagic conversion of streptococcal pneumonia. He required frequent resuscitation throughout the evening with aggressive fluid and vasopressor support. Dialogue was initiated with the family who wished to move the patient to hospice care. He died early the next morning. No ECMO was initiated.

Discussion

The patient in this case illustrates the necessity for the development of additional strategies for patients with severe respiratory compromise. He suffered from overwhelming sepsis because of complications from streptococcal pneumonia. His condition worsened to the point where his diagnosis shifted from that of relatively simple respiratory failure to that of a patient with septic shock complicated by multiorgan failure. Additionally, given the degree of hypoxemia, he easily qualified as having a concurrent diagnosis of acute respiratory distress syndrome (ARDS).

ARDS is a disease of inflammation that occurs as a result of primary lung disease or secondary to systemic inflammation caused by other disease processes such as sepsis. It occurs as a result of an inflammatory process involving the alveolar-capillary membrane, resulting in intrapulmonary shunting with hypoxemia and pulmonary hypertension.[1] The inflammatory process leads to increased pulmonary capillary permeability with the net result of noncardiogenic pulmonary edema that is classically seen as a result of either primary lung injury or as a secondary insult from the same concurrent or precipitating systemic illness. Because of the ventilation-perfusion mismatch, intrapulmonary shunting occurs as a result of increased vasodilation in nonventilated portions of the lung and vasoconstriction in ventilated areas.[1] Thus, the patient experiences a 2-fold insult of hypoxemia and pulmonary hypertension.

To qualify the severity of ARDS, providers measure the PaO_2/FiO_2 (P/F) ratio.[2] Mild ARDS is identified in those patients with a P/F ratio of < 300 mm Hg, whereas those with more severe ARDS are identified with a P/F < 100 mm Hg.[3] Despite these definitions, few successful mechanical and pharmacological therapies exist to treat this significant illness.

As outlined in a variety of research, studies, and protocols, the treatment of ARDS has not only focused on treating the initial insult, but also on developing strategies for the most efficient form of oxygenation and ventilation. Protocols outlined in guidelines such as the Acute Respiratory Distress Syndrome Clinical Network have provided mechanical ventilator strategies with specific recommendations for respiratory rate, expiratory time, tidal volume, positive end-expiratory pressure, and FiO_2.[4,5] However, in critically ill patients with severe cases of ARDS, other measures are being explored to supplement current treatment modalities. In the current case, the P/F ratio was 58 mm Hg, and as noted with the degree of illness, further strategies were looked upon as "salvage" therapy in an attempt to prevent death in this patient. In the remainder of this article, we will address 3 therapies that have recently gained popularity including nitric oxide, nebulized prostaglandins (epoprostenol), and ECMO.

Inhaled nitric oxide (iNO) is a potent vasodilator that is routinely used in patients to improve oxygenation and reduce pulmonary vascular resistance in the setting of elevated pulmonary resistance and pulmonary hypertension seen in the setting of ARDS.[6,7] Although iNO is effective at improving oxygenation, there are no data that demonstrate a reduction in mortality. In a recent meta-analysis, multiple trials were analyzed, and an overall benefit was only transiently seen for the first 24 hours. At 48 and 72 hours, there was no significant effect on morbidity and mortality.[1] Additionally, researchers also identified that the use of iNO may lead to increased rates of acute renal failure. Lastly, the studies analyzed have significant confounding factors and were limited in the scope of each trial because of the patient population.[1] At this point, the efficacy of iNO in acutely ill patients remains debatable with limited use, but in patients who are being transitioned to other significant therapies, it continues to be used as a bridge when other modalities have previously failed.

In recent years, because other pharmacological interventions such as iNO have shown limited benefit, other therapeutic options have been identified that compliment previously tested medications including beta-adrenergic agonists, low-dose