

Received: 2020.04.06

Accepted: 2020.06.24

Available online: 2020.07.14

Published: 2020.08.25

A Novel Ventilatory Technique in Refractory Hypoxemic Respiratory Failure Secondary to Therapeutic Thoracentesis and Paracentesis

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Authors' Contribution:

Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

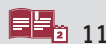
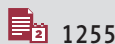
Manuscript Preparation E

Literature Search F

Funds Collection G

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Patient: Male, 61-year-old
Final Diagnosis: Hypoxemic respiratory failure • reperfusion pulmonary edema
Symptoms: Respiratory deterioration
Medication: —
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Unusual or unexpected effect of treatment**Background:** MetaNeb® is a respiratory therapy modality that aims to effect clearance of airway secretions through chest physiotherapy. It typically is used in critically ill patients with bronchiectasis or copious secretions. However, it also expands lungs through a continuous positive expiratory pressure and continuous high-frequency oscillation, which has the benefit of increasing lung recruitment and improving oxygenation.**Case Report:** A 61-year-old male who had re-expansion pulmonary edema following a paracentesis and thoracentesis for cirrhosis, which caused a large unilateral pleural effusion. He required intubation and his hypoxemia was refractory to standard maximum ventilatory measures. A trial of continuous MetaNeb® acted as a noninvasive extracorporeal membrane oxygenation method, dramatically improving oxygenation and hypoxemia, normalizing the patient's blood gas, and thus stabilizing him.**Conclusions:** MetaNeb® could potentially be used in other community hospitals that lack the capability for advanced ventilatory modes or in patients who are too unstable for transfer.**MeSH Keywords:** Hypoxia, Brain • Pulmonary Edema • Ventilators, Mechanical**Full-text PDF:** <https://www.amjcaserep.com/abstract/index/idArt/924862>

Background

Reexpansion pulmonary edema (REPE) is a possible complication of both paracentesis and thoracentesis, although most patients do not receive both procedures in rapid succession. REPE is described in the literature as a rare but severe clinical condition with incidence of 0.2% to 14% and mortality up to 20% due to rapid reinflation of collapsed lung tissue after treatment of atelectasis, pleural effusion, or pneumothorax [1–3]. The first reported case of REPE was described in 1958 by Carlson in a patient with whole-lung collapse secondary to pneumothorax [2]. Risk factors include young age, female sex, greater degree and duration of lung collapse, reexpansion of the lung in less than 10 minutes, and use of negative pressure during treatment with evacuation of more than 2000 mL [2].

Clinical features of REPE range from asymptomatic to respiratory failure and circulatory collapse. Early diagnosis and rapid initiation of supportive care is critical to prevent mortality.

Typically, patients develop severe coughing or chest tightness during or within a few hours post-procedure, sometimes producing frothy sputum. Other features may include dyspnea, tachypnea, tachycardia, fever, hypotension, cyanosis, nausea, or vomiting. Symptoms typically progress over a period of 24 to 48 hours, and chest x-ray shows pulmonary edema throughout the ipsilateral lung. It may also develop in the contralateral lung [2,4]. Recovery is complete in 48 hours if the patient does not die.

Treatment is supportive with positive-pressure mechanical ventilation to reexpand lung tissue and reduce shunting by increasing alveolar recruitment [3]. Vasopressors, especially inotropes, and diuretics can be used as adjunctive therapies. Patients with cirrhosis are typically more prone to baseline hypotension due to relative intravascular depletion. Inotropes such as dobutamine can be helpful in the setting of underlying heart failure [2,5]. In addition, cirrhosis can result in cardiovascular complications including cirrhotic cardiomyopathy, portopulmonary hypertension, and hepatopulmonary syndrome, which may manifest or worsen with hemodynamic stress associated with procedures such as thoracentesis and paracentesis [6].

Few case reports describe acute onset of pulmonary edema or cardiac arrest in patients after paracentesis or thoracentesis, especially in cirrhosis. There are no other described cases of patients with REPE following both procedures. Recommendations to prevent REPE include consideration of the duration of pleural effusion, underlying lung disease, pleural pressure, and symptoms during thoracentesis. Consensus is that less than 1500 mL should be removed unless pleural pressures are monitored [3].

Case Report

A 61-year-old male weighing 104 kg presented with acute onset of dyspnea with cirrhosis and ascites. His comorbidities included diabetes, hypertension, and chronic kidney disease. The patient's cirrhosis was due to nonalcoholic steatohepatitis. He had mild diastolic dysfunction with a preserved ejection fraction on echocardiogram. He was initially started on bilevel positive airway pressure (BiPAP) with some improvement with settings of a peak inspiratory pressure (PIP) of 12 and a positive end-expiratory pressure (PEEP) of 5. His initial blood gas in the Emergency Department showed a pH of 7.399, partial pressure of carbon dioxide ($p\text{CO}_2$) of 33.4, and partial pressure of oxygen ($p\text{O}_2$) of 63. The patient received diuresis and was started on albumin infusions. The interventional radiology service performed a thoracentesis of 1850 mL pleural fluid as well as a paracentesis of 1350 mL of ascitic fluid in immediate succession, for a total of 3200 mL removed. Immediately post-procedure, he developed worsening respiratory distress. At that time the patient's pH was 7.372, $p\text{CO}_2$ 40.6, and $p\text{O}_2$ 49 on a nonrebreather mask. He again required BiPAP. Computed tomography scan of the chest was obtained and showed severe extensive bilateral reperfusion pulmonary edema. The patient developed rapidly worsening hypoxia with oxygen saturations into the 80s. Of note, his effusion was favored to be transudative based on an LDH pleural fluid of 86, pleural protein level of <2.0, and a serum protein of 7.0.

He was subsequently intubated and placed on a Puritan Bennett 840 Ventilator, but had persistent hypoxemia. He was maintained on assist control ventilation with a tidal volume of 400, rate of 14, PEEP of 10 cmH_2O , and FiO_2 of 100%. Endotracheal tube suctioning improved saturations briefly but he continued to have copious pink frothy secretions. His oxygen saturations remained in the 70s and low 80s. He was also profoundly hypotensive, requiring a maximal rate of intravenous norepinephrine, vasopressin, and epinephrine. He deteriorated into pulseless electrical activity twice with quick return of spontaneous circulation after epinephrine pushes. Dopamine and Neo-Synephrine drips were started, with oxygen saturations in the 60s to 70s post-resuscitation. Tidal volume was uptitrated to 480 mL with a rate of 18. Pressure control ventilation was attempted at 22 cmH_2O to no further avail. His arterial blood gas revealed a pH of 7.185, $p\text{CO}_2$ of 48, and $p\text{O}_2$ of 48 on 100% fraction of inspired oxygen (FiO_2). Diuresis were not possible due to the severity of the patient's circulatory collapse.

Endotracheal suctioning revealed copious pulmonary edema, and 900 mL was suctioned over 2 hours. Vecuronium was attempted to see if oxygenation would improve, which did not correct the patient's hypoxemia. With the help of Respiratory Therapy, the MetaNeb® was placed on high flow to see if it would decrease secretions and improve oxygenation. Once

the patient was connected, his oxygenation immediately improved to the high 80s and ultimately to 100%. His blood gas improved while on the MetaNeb® continuously for 5.5 hours, normalizing to a pH of 7.492, pCO₂ of 29.1, and pO₂ of 134. He had no further desaturations and his pressors were titrated down. When the MetaNeb® was shut off, the patient quickly desaturated into the 70s and 80s, requiring manual bagging with another episode of cardiac arrest. Unfortunately, the patient had suffered an anoxic brain injury due to the hours of prolonged hypoxia and he continued to require substantial pressor support. The family opted for compassionate extubation and he expired quickly.

Discussion

MetaNeb® is a respiratory therapy modality that aims to effect clearance of airway secretions through chest physiotherapy. It typically is used in critically ill patients with bronchiectasis or copious secretions. However, it also expands lungs through a continuous positive expiratory pressure and continuous high-frequency oscillation over 10-minute cycles, which has the benefit of increasing lung recruitment and improving oxygenation [7]. Its use has been described in case reports to mobilize secretions for sputum culture in patients with chronic obstructive pulmonary disease and in short intervals for critically ill pediatric patients [8–11]. It has also been used as an aggressive pulmonary toilet mechanism in postoperative

patients. However, its continuous use has not been described in adults on a mechanical ventilator or as an effective treatment modality for pulmonary edema.

In our community hospital setting without advanced ventilatory modes or the immediate availability of an extracorporeal membrane oxygenation (ECMO) team, continuous MetaNeb® therapy can treat hypoxemia that is refractory to standard ventilatory modes in patients who are too unstable for transfer, which may ultimately reduce risk of anoxic brain injury. The MetaNeb® ultimately acts as an external and noninvasive type of ECMO, dramatically and rapidly improving oxygenation. It can help reduce secretions and work in conjunction with diuresis to stabilize patients onto traditional ventilatory modes. Further investigation is warranted of the potential for use of MetaNeb® as an adjunct therapy for critically ill patients on mechanical ventilation and refractory hypoxemia due to alveolar gas mismatch.

Conclusions

Continuous MetaNeb® use is an innovative adjunct to traditional ventilatory modalities. MetaNeb® should be considered in community hospital patients with refractory hypoxemia who are not stable for transport and do not have access to advanced ventilatory modes. Further investigation is needed to explore its potential to improve refractory hypoxemia.

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