ABSTRACT

Severe Influenza and superimposed Streptococcal Pneumonia-Induced ARDS in a Young Patient: Escalation to ECMO Support

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INTRODUCTION

ARDS (acute respiratory distress syndrome) is a critical condition characterized by severe hypoxemia and impaired gas exchange, often requiring advanced therapeutic interventions.

This abstract highlight a rare and life-threatening case of ARDS induced by severe influenza virus infection and superimposed Streptococcus pneumonia.

CASE DESCRIPTION

A 44-year-old male with known history of prior opiate abuse on Suboxone, history of anxiety, obesity presented to hospital for progressive shortness of breath, cough symptoms of 3 to 4 days duration. He started having initial upper respiratory infection symptoms followed by symptoms of cough, chest tightness, mucus expectoration associated with fever, chills and bodyaches. In ED, patient was hypoxic on room air due to which was placed on nonrebreather mask subsequently placed on BiPAP due to increased work of breathing and hypoxia. On physical examination, bilateral coarse breath sounds, rhonchorous, reduced bilaterally.

Initial findings include he tested positive for influenza A. Initial ABG on arrival 7.24/38/76/16 on 100% BiPAP. Urine drug screens were positive for amphetamines and benzos. Lactate was elevated to 6.49, down trended to 4.2 s/p Sepsis bolus. Initial Creatinine was 2.83 down trended to 2.61, Potassium 6.6 >6.4, Bicarbonate of 18. Streptococcal pneumoniae antigen tested positive. Chest x-ray on admission revealed left upper and lower lobe pneumonia. Patients started on Steroids, Ceftriaxone and doxycycline along with Tamiflu.

HOSPITAL COURSE:

MET was called next morning for severe hypoxia, tachypnea, tachycardia despite being on BiPAP due to which patient was intubated. X-ray was done which revealed worsening right infiltrates which were clear on initial X-ray, now showing Bilateral infiltrates. He was still hypoxic, so PEEP was increased to 12 and FiO2 to 100%. His X-ray was suggestive of ARDS. He had persistent hyperkalemia and metabolic acidosis due to which he was started on CVVHD which was stopped on day 2 due to improved in kidney function. His hypoxia continued to worsen, and his condition deteriorated within few hours due to proning and Flolan was started. Despite FiO2 of 100%, low TV and PEEP of 14 along with proning and inhaled vasodilator, patient's condition continued to worsen due to which VV ECMO was started through right femoral and left femoral vein due to difficult wire placement in right IJ. VV-ECMO was changed to right IJ-right femoral vein to reduce recirculation and better lung rest. ABG 7.49/42/88/25 on VV ECMO 70% and ventilator PRVC 14/320/40/12. ECMO settings and FiO2 on vent were reduced based on daily ABGs with significant improvement. ECMO was decannulated on day 6 and patient was liberated from mechanical ventilation transitioned to nasal cannula on day 7. Repeat Chest X-ray revealed significant improvements.

DISCUSSION:

ARDS due to any injury especially infections like flu and bacterial infections lead to acute lung injury with increased hydrostatic extravascular lung water, reduced compliance and severe hypoxemia.

Severe forms of influenza infection superimposed bacterial pneumonia complicates the infection increasing risk of acute respiratory distress syndrome (ARDS) increasing need for mechanical ventilation and subsequent need for Extracorporeal membrane ventilation in patient not benefiting from traditional management. Mechanical ventilation remains the most important aspect of managing patients with ARDS with high positive end expiratory pressures and low tidal volumes. In the first four hours, patient's clinical response, gas exchange, plateau pressure (<30cc H2O important target) and driving pressures are monitored to adjust ventilator settings. Other ventilator setting modes can be tried in patients not tolerating volume-limited

LTVV (unable to achieve a plateau pressure <30mm H2O, ventilator desynchrony) including pressure-limited modes, increased inspiratory to expiratory ratio (inverse ratio ventilation) which prolongs the inspiratory time improving oxygenation by allowing regions of lung to open and participate in gas exchange. High PEEP maximizes alveolar recruitment. A successful response is considered when PaO2/FiO2 ration maintains or increases to >150mm Hg. In patients with refractory hypoxemia (partial arterial pressure of oxygen/fraction of inspired oxygen ratio <150) and/or plateau pressure >30cm H20 despite low tidal volume ventilation and appropriate PEEP, and other supportive measures, prone ventilation and pharmacotherapy like neuromuscular blockers and pulmonary vasodilators are considered. Contraindications to Prone position include active bleeding, multiple fractures, spinal instability, raised intracranial pressure >30mmHg or tracheal surgery or sternotomy within 2 weeks. When Proning is not feasible or has failed, early ECMO consideration is done.

Severe, acute ARDS or refractory ARDS may benefit from VV-ECMO. ECMO is a form of extracorporeal life support which provides lung rest as oxygen exchange and carbon dioxide removal occurs through external circuit allowing lungs to recover. ECMO is weaned off when patients have improved lung compliance (tidal volume 6mL/kg predicted body weight with plateau airway pressure <30cc H20, improving radiograph appearance and adequate oxygenation at low fraction of inspired oxygen.

ECMO lead to improved in this which was started due to worsening hypoxemia despite high PEEP, low TV ventilation, Prone positional and Flolan (inhaled vasodilator).

CONCLUSION:

The synergistic effect of Flu with superimposed bacterial infection can rapidly worsen respiratory function, overwhelming the lung's ability to oxygenate blood, and leading to the need for extracorporeal membrane oxygenation. ECMO, an advanced life support technique, provides temporary circulatory and respiratory support to patients with refractory respiratory failure.

ECMO provided significant improvement In ARDS in this patient by allowing lungs to rest and recover.