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ECMO in young male with Leptospiral ARDS

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Abstract
Leptospirosis is a zoonotic disease associated with mild to severe pulmonary complications. On rare occasions, ARDS can be secondary to tropical disease. Accordingly, a history should include travel to endemic regions. Acute respiratory distress syndrome (ARDS), characterized by hypoxemic respiratory failure, is associated with a high mortality of and is precipitated by both direct and indirect pulmonary insults. Treatment is largely supportive, consisting of lung protective ventilation and occasionally requires Extra-corporeal membrane oxygenation (ECMO) and thereby necessitating Intensive Care Unit (ICU) admission. We describe a case of a 18-year-old male with undiagnosed leptospirosis, presenting with fever and severe hypoxemic respiratory failure, returning from a holiday. He was intubated and received lung protective ventilation followed by prone ventilation which failed. His condition improved after ECMO and antibiotic added empirically. This case illustrates the rare Complication of ARDS from leptospirosis, which may need ECMO and the importance of taking a travel history.

Keywords: Acute respiratory distress syndrome (ARDS), Extra-corporeal membrane oxygenation (ECMO), Leptospirosis, Microscopic agglutination testing (MAT)

Introduction
Leptospirosis is a zoonotic disease most common in the tropics and typically associated with mild to severe pulmonary complications. The mortality rate in leptospirosis cases with severe pulmonary hemorrhagic syndrome is 30–60% even with adequate treatment [1, 2]. Malaria, dengue, and chikungunya share some common clinical features and similar endemic pattern with leptospirosis [3]. Transmission to humans is most commonly environmental *via* contact with water or damp soil contaminated with leptospires, but may also occur from direct contact with urine or blood from an infected or colonized animal. Organisms then spread to the bloodstream and multiply, and hematogenous dissemination throughout the body occurs, with potential to affect nearly every organ system due to the ability of the spirochetes to easily cross tissue barriers before the host antibody response clears them from the blood [4].

Case presentation
A 18-year-old male with no previous medical comorbidities was brought to the Emergency Department (ED) with complaint of fever, one episode of hemoptysis and increasing tachypnea and dyspnea over the previous day. His mother reported that the patient went to forest trip with friends. He has never used any medications. Immediately upon returning, the patient saw his primary care physician, and was given paracetamol. In the following days, the patient's condition worsened and he presented to the ED with the following vital signs: blood pressure of 110/70mmHg, heart rate (HR) of 124 beats/min, respiratory rate (RR) of 45 breaths/minute, temperature of 38.4 degrees Celsius, and oxygen saturation of 86%. Glasgow Coma Scale (GCS) was 15/15, with conjunctival suffusion. He was in obvious respiratory distress with accessory muscle use. A portable chest radiograph demonstrated bilateral opacities. The patient was placed on a nonrebreather (FiO2 of 1), but his work of breathing did not substantially improve, and he remained hypoxemic. Therefore, the patient was put on non-invasive ventilation (NIV) in the ED and an arterial line was inserted. ED bloodwork revealed a pO2 of 55mmHg on 100% oxygen, but normal electrolytes, renal function, liver enzymes, and coagulation parameters. Following ICU transfer, the patient was diagnosed with ARDS due to his severely depressed PaO2/FiO2 ratio (110 upon ICU admission), bilateral opacities, and no signs that would suggest cardiac dysfunction (i.e., normal blood pressure, no peripheral edema, and normal electrocardiogram).

He was intubated and ventilated with a standardized ARDS Net protocol that focused on low tidal volume (6mL/kg of ideal body weight and a target pH > 7.25), plus a positive end-expiratory pressure (PEEP) increased to 12mmHg. Despite intubation PaO2/FiO2 ratio remained below 100 prone position tried but patient persistently remained hypoxic with FiO2 1.0.ECMO team called on same day and patient cannulated and put on ECMO. While the etiology for ARDS was unclear, it was presumed infectious in origin, given history of fever and travel. Blood and urine cultures were performed. Transthoracic echocardiogram revealed normal biventricular function, with no vegetation or valvular regurgitation. Patient started on broad-spectrum antimicrobials (piperacillin-tazobactam and doxycycline). Initial blood and urine grew no pathogens. Malaria thick and thin smears were performed several times and were negative. Dengue serology was also negative, as was human immunodeficiency virus (HIV) and hepatitis. Patient had blood stained secretions, next day CXR shown left Lung collapse. Bronchoscopy shown multiple clots which were removed. Post bronchoscopy CXR became clear.

Patient remained on ECMO with High FiO2 and high flow rate. Antibiotics modified to Meropenem, Doxycycline continued. We maintained the ECMO blood flow at >4L/min during the first 3 days of ECMO to maintain oxygenation. After PaO2 improved, we decreased the ECMO blood flow gradually to 1.5L/min when peripheral capillary oxygen saturation (SpO2) was over 90%. During V-V ECMO the mechanical ventilation was set at the lung rest setting, which consisted of a PEEP of 10cm of water, and FIO2 of 0.21. The patient was systemically heparinized to maintain an activated partial thromboplastin time of 70–80 seconds. He required high dose sedation with midazolam and fentanyl and intermittently muscle relaxant atracurium. His creatinine jumped gradually from 0.8 to 2.4 and later normalized. On 5th day flow rate started reducing, FiO2 requirement started declining drastically. Day 9, patient weaned off from ECMO, on day 10 extubated and Decannulated on same day. On Day 11 patient shifted to ward and on Day 12 patient discharged. First Leptospira IgM was negative which turned out to be positive when repeated after 1 week. Diagnosis was further confirmed with microscopic agglutination testing (MAT).

Discussion

Leptospirosis is the most common zoonotic infection in the world. It is easily transmitted from infected animals through their urine, either directly or through infected soil or water. [5, 6] It can cause a self-limiting influenza-like illness or a much more serious disease. It is known as Weil disease, and it can progress to multiorgan failure with the potential for death. Definitive diagnosis of leptospirosis requires recovery of leptospires either by culture or by immunohistochemical staining. Serology can also be performed using MAT or IgM-detection. Regardless of the method used, results can take days to weeks, and, therefore, if there is a high degree of suspicion, patients should receive empiric treatment. There is a paucity of evidence regarding optimal antimicrobials for leptospirosis, but sources recommend oral doxycycline for prophylaxis/mild disease [7, 8] and injection doxycycline, ampicillin and penicillin G for severe disease [9]. Ceftriaxone is a suitable alternative for treatment of severe disease and in patients with a penicillin allergy [10]. The mechanism by which leptospirosis triggered ARDS is unclear. Our patient was diagnosed with ARDS but assumed to have a typical bacterial etiology. Leptospirosis considered because of history of forest visit, hemorrhagic conjunctivitis and pulmonary hemorrhage after admission led to empiric therapy.

The pathophysiology of pulmonary injury in leptospirosis is poorly understood. The pulmonary injury may be caused by an undefined leptospiral toxin that induces endothelial damage in pulmonary capillaries, or by host immune responses [11, 12, 13]. Methylprednisolone has been used for treatment of pulmonary hemorrhage on the basis of the pathogenetic mechanism of lung injury; however, no significant mortality benefit has been observed in patients already on mechanical ventilation [14] we avoided use of Methylprednisolone. Trivedi *et al*. evaluated the efficacy of cyclophosphamide and plasma exchange in patients with leptospiral pulmonary hemorrhage. However, patients with severe disease may not tolerate the transient hypoxemia associated with plasma exchange [15]. Alternatively, V-V ECMO, which supports pulmonary function and minimizes the damage induced by mechanical ventilation, could be useful until the lungs recover. Liao *et al*. have reported the successful use of VV ECMO for the treatment of severe respiratory failure due to alveolar bleeding and acute respiratory distress syndrome [15].

The cause of severe respiratory failure in the present case was massive alveolar bleeding and non cardiogenic pulmonary edema. Marked alveolar bleeding was observed during bronchoscopy. In view of refractory hypoxeamia despite prone position patient was put on V-V ECMO. If a patient has flu-like symptoms and pulmonary hemorrhge, it is important to rule out leptospirosis as a differential diagnosis. The patient should be carefully monitored in the intensive care unit as respiratory distress may progress rapidly. In addition, V-V ECMO therapy should be considered early in case of refractory hypoxemia because it could be life threatening.

Conclusion

Our inference is, patient with leptospirosis-associated pulmonary hemorrhage and ARDS could be successfully treated by using V-V ECMO with careful anticoagulation therapy and survival of patients with leptospirosis could be improved.

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