

Successful use of extracorporeal membrane oxygenation in a patient with transfusion-related acute lung injury

Jarrold Mosier, Terence O'Keeffe, Holly Graham, Peter M. Rhee

Abstract

Objective: Transfusion-related acute lung injury (TRALI) is a relatively uncommon complication in patients who undergo plasma-containing blood product transfusion. Despite the cross matching process, TRALI remains the most common cause of mortality associated with transfusion. We describe a case of TRALI in a 24-year-old trauma patient who was successfully cared for on extracorporeal membrane oxygenation (ECMO).

Design: Chart review.

Setting: Academic medical center with Level 1 Trauma.

Patients and participants: Single case from a busy urban trauma center.

Interventions: Extracorporeal membrane oxygenation.

Conclusion: ECMO has only rarely been successfully used in patients with TRALI; to our knowledge, ours is the first successful report of its use in a trauma patient. Its use in maintaining gas exchange in trauma patients with TRALI should be further investigated.

Key words: TRALI, ECMO, trauma, acute lung injury, transfusion reaction, respiratory failure.

Introduction

Transfusion-related acute lung injury (TRALI) is a rare and potentially fatal complication of blood product transfusion. However, it represents the most common cause of transfusion-related mortality, with an incidence of around 1:5,000. (1-4) Hypoxia and hypotension are the major clinical consequences, with the extreme end of the

clinical spectrum characterized by acute pulmonary edema and profound shock even in patients with baseline normal heart function. Treatment involves respiratory support (to maintain oxygenation) and fluids (to maintain circulatory volume).

Most patients with TRALI require only mild supplemental oxygen or noninvasive ventilation. But severe cases can require intubation and positive-pressure ventilation, often with less conventional modes of ventilation. We know of only 2 prior reports of successful resuscitation with extracorporeal membrane oxygenation (ECMO) in patients with TRALI: in 2003, Nouraei et al described a 4-year-old girl who developed TRALI after elective cardiac surgery; more recently, in 2008, Lee et al described a 22-year-old obstetric patient. (5,6) In this case report, we describe the first successful use of ECMO for TRALI in a trauma patient with severe pulmonary failure after

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hemorrhagic shock and resuscitation.

Case report

A previously healthy 24-year-old man was struck by a car while acting as a Good Samaritan on the side of the road. On presentation to the Emergency Department, his Glasgow Coma Scale (GCS) was 15; he was alert and oriented, with left flank pain and nausea. His initial vital signs were as follows: blood pressure 157/85, heart rate 133, and oxygen saturation 97% on room air. He had no relevant prior medical history, took no regular medications, and had no allergies. A FAST exam was not performed initially, however further workup by computed tomography (CT) of the chest, abdomen, and pelvis revealed a splenic laceration and shattered left kidney (grade IV/V) (**Figure 1**). The initial management plan consisted of angioembolization of his renal laceration, but during this procedure, and before embolization could be performed, the patient became hemodynamically unstable and was taken emergently to the operating room for a nephrectomy.

A total nephrectomy was rapidly performed, with intra-operative exploration revealing a grade V renal injury and no other significant injuries. Intra-operatively, the patient received 10 units of packed red blood cells (PRBCs), 12 units of fresh frozen plasma (FFP), and 2 units of platelets. Shortly after we closed the abdomen, the patient became profoundly hypoxic, with profuse frothy sputum coming from his endotracheal tube (ETT). We administered high oxygen supplementation and positive end-expiratory pressure (PEEP) ventilation. A chest X-ray showed patchy bilateral infiltrates (**Figure 2**). He maintained a PaO₂ under 20 mmHg for more than 1 hour, despite maximal ventilatory support. Results of transesophageal echocardiography were consistent with hypovolemia. We performed rescue ventilation by starting ECMO, which included placement of a third catheter to provide perfusion to the right leg.

In addition to commencing ECMO, the patient was also placed on airway pressure release ventilation (APRV), with a peak pressure of 50 mmHg, 100% FiO₂, and 40 ppm of inhaled nitric oxide (**Figure 3**). Initially, he required vasopressors for adequate pump flow; however, his oxygenation quickly improved.

Over the next 48 hours, we used vasodilators to avoid increased pulmonary blood flow, as any such increase would be deleterious by bypassing the ECMO oxygenation mechanism. As the patient's pulmonary edema and acute respiratory distress syndrome (ARDS) resolved, he was weaned off ECMO and decannulated on postoperative day 3. He underwent a tracheostomy on day 7 because of poor mental status, but his respiratory status continued to improve and he was weaned off the ventilator on postoperative day 12.

His hospital course was complicated by a ventilator-associated pneumonia caused by *Pseudomonas* and *Enterobacter* species; he was successfully treated with an 8-day course of intravenous Zosyn, followed by oral ciprofloxacin. He recovered quickly and his tracheostomy tube was removed on postoperative day 19. He was discharged home, with no morbidity, on postoperative day 23. At his first follow-up visit 4 weeks later, he had completely recovered. Results of his 6-month pulmonary function tests were normal. Hematologic test results, which demonstrated anti-HLA antibodies in both the patient and in 1 donor's blood, supported the diagnosis of TRALI. In addition, we highly suspect the presence of a human neutrophil antibody, which was not tested for by the blood bank.

Discussion

The diagnosis of TRALI requires acute-onset hypoxia within 6 hours after blood product transfusion, a PaO₂/FiO₂ ratio under 300 mmHg, no left atrial hypertension, a wedge pressure under 18 mmHg, and bilateral patchy pulmonary edema seen on X-ray. (1,3,7) Making the diagnosis can be difficult if the patient has other risk factors for ARDS; the telling clue may be the temporal relationship to the transfusion. Blood products containing plasma have the highest risk of causing TRALI, because they carry the instigating antibodies. The order of risk is as follows: platelets derived from whole blood, followed by FFP, PRBCs, whole blood, platelets, cryoprecipitate, and intravenous immunoglobulin (IVIG). (1-3,8)

Silliman et al described the 2 leading proposed mechanisms of TRALI in an immunocompetent patient. (9) One proposed

mechanism is that transfusion of blood products containing antibodies to recipient leukocytes adhere to the pulmonary endothelium, leading to activation and an uncoordinated release of inflammatory processes, which causes capillary leak and acute lung injury. Alternatively, according to the other proposed mechanism, infusion of blood products containing leukocytes for which the recipient has an antibody leads to the same cascade. The end result is a massive activation of leukocytes sequestered in the pulmonary endothelium with no stimulus to coordinate the activity. Interestingly, transfusion products derived from multiparous women have the highest risk of producing TRALI, given such women's higher likelihood of developing various anti-HLA or anti-granulocyte antibodies after pregnancy. (7,8) Clinically, the effects can range from mild hypoxia (requiring minimal supplemental oxygen) to massive protein-laden frothy sputum, hypovolemia, profound hypoxia, and shock (requiring full ventilatory support).

TRALI, like ARDS, can require profound ventilatory support and rescue techniques. High PEEP ventilation, which may be needed to stent open atelectatic alveoli, can prevent harmful shear stress on non-diseased alveoli when high peak pressures are required. Once shear stress reaches a certain threshold, volutrauma and barotrauma ensue, drastically worsening the mortality rate associated with ARDS. Lung protective strategies have greatly reduced the mortality rate associated with ventilator use in ARDS patients, but such strategies have their limits. Once a certain threshold is reached, rescue strategies must be started. APRV, an initial ventilator strategy, can provide improved oxygenation in the setting of controlled higher peak and plateau airway pressures. Inhaled nitric oxide can be used to decrease the Ventilation/Perfusion mismatch by dilating blood vessels adjacent to well-ventilated alveoli.

ECMO was originally developed to provide a low-flow lung bypass for patients with ARDS and for newborns in respiratory distress. The early studies, in the 1970s, showed very disappointing survival rates of about 10%. (10) But more recent studies in the 1990s have showed survival rates nearing 50% in adults with ARDS. (11) Michaels et al studied ECMO as an early rescue strategy in patients with severe pulmonary failure (defined as P/F ratio under 100, despite inverse ratio ventilation and

optimal PEEP ventilation) and found a survival benefit, with an odds ratio of 7.2. (12) It may be, in patients with TRALI, that ECMO should be considered as a rescue strategy and initiated early in the clinical course. Though a form of ARDS, TRALI has a shortened clinical course, typically around 72 hours, which is drastically different from the protracted recovery time of patients whose ARDS has other causes.

In our patient, early cannulation and aggressive use of other rescue strategies (APRV and inhaled nitric oxide) provided acceptable oxygenation and ventilation, while we resolved the state of shock induced by TRALI. Our patient was weaned and decannulated after several days, then adequately supported with APRV until his complete respiratory recovery. On his follow-up visit at 4 weeks, then again at 6 weeks, he had no disabilities and enjoyed a complete neurologic, cardiopulmonary, and renal recovery.

Figure 1. Representative coronal reconstruction slice of the computed tomography (CT) scan, obtained on our patient's admission, demonstrating the shattered left kidney

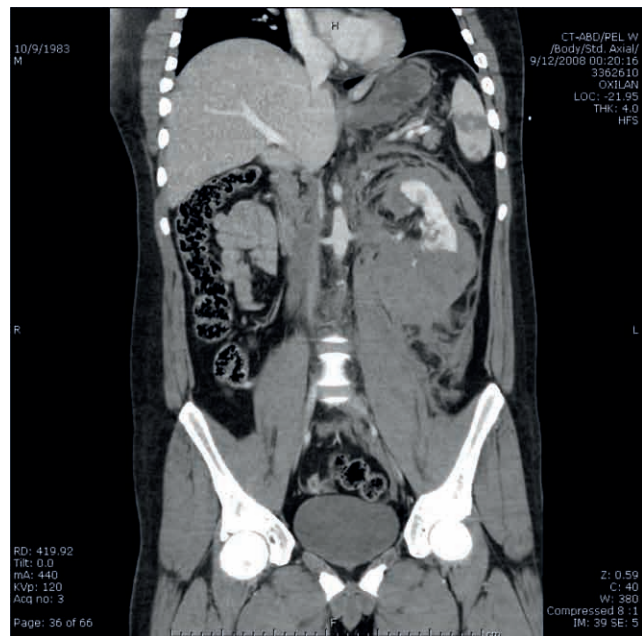


Figure 2. Chest X-rays obtained during our patient's initial trauma evaluation (upper) and at the onset of respiratory failure (bottom), demonstrating the sudden onset of pulmonary edema

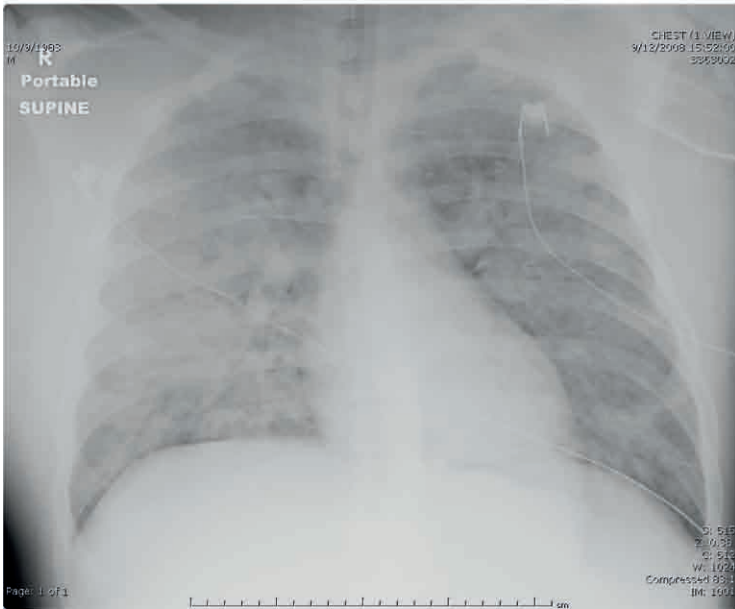
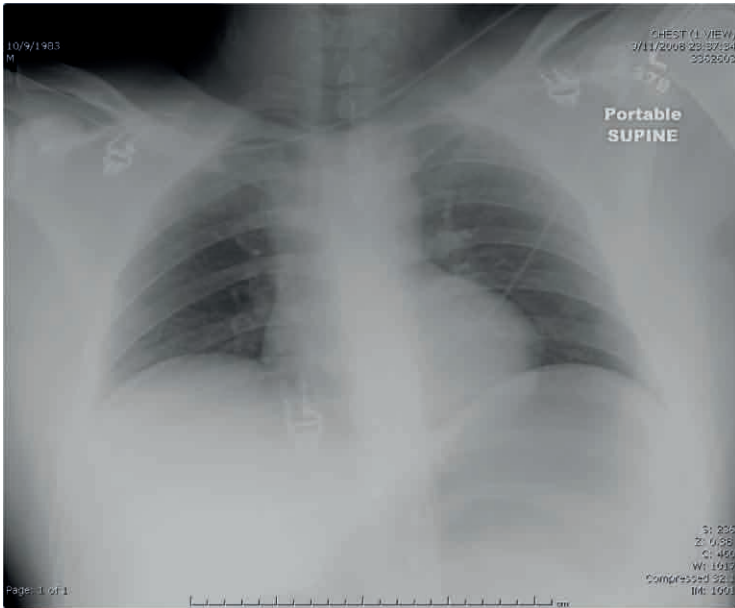
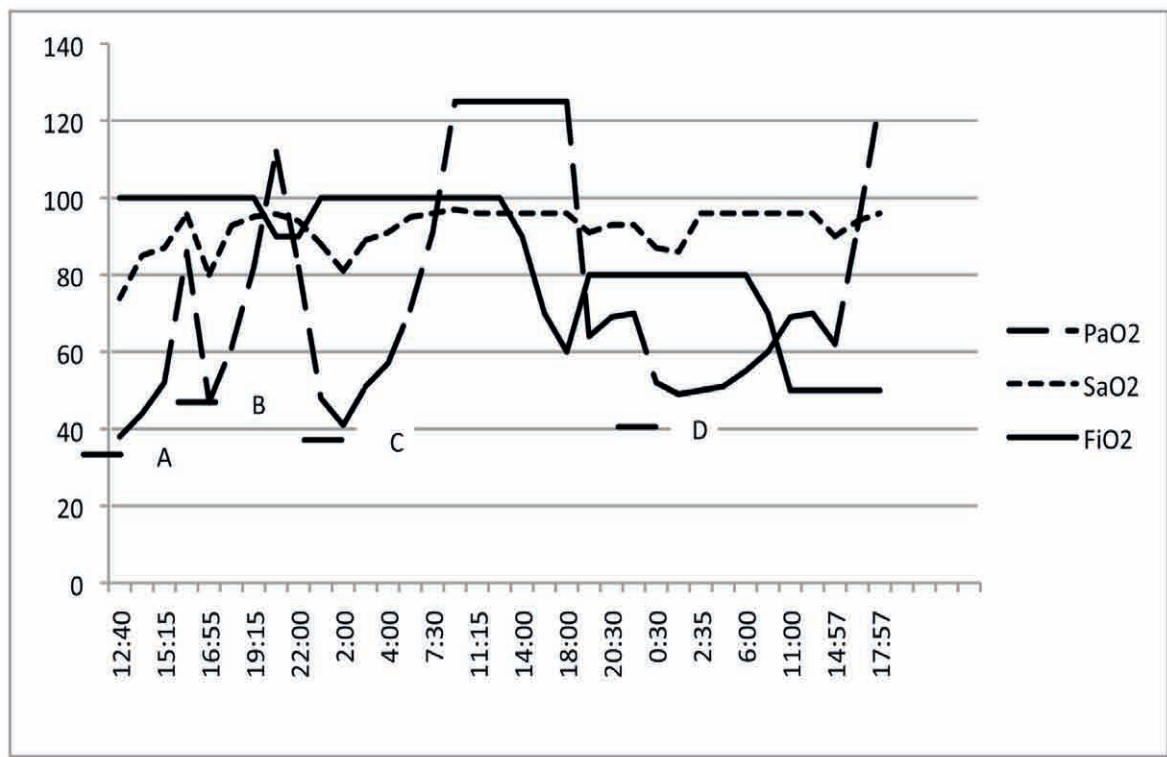


Figure 3. Arterial blood gas trends



Legend: Before extracorporeal membrane oxygenation (ECMO) (A); after cannulation (B). Even on ECMO, early attempts to wean our patient off positive end-expiratory pressure (PEEP) ventilation and FiO2 resulted in drastic decreases in PaO2 and SaO2. To maintain oxygenation, ECMO and inhaled nitric oxide (C) were required in the first 48 hours. After decannulation (D), our patient required higher FiO2 and PEEP ventilation; however, he was weaned over several days (data not shown)

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