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Extracorporeal Membrane Oxygenation Successfully Resuscitated a Patient with Severe Transfusion-related Acute Lung Injury

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Transfusion-related acute lung injury (TRALI) is a serious life-threatening complication of blood transfusion, and the available treatments are mainly supportive. Extracorporeal membrane oxygenation (ECMO) is currently being widely used in intensive care units for treating patients with respiratory failure, but is rare for TRALI. We report a 20-year-old man who sustained a crushing injury with close fracture of the right clavicle and scapula, the left pubic ramis and urethral disruption. TRALI developed within 72 h after a massive transfusion of blood products. The patient remained profoundly hypoxic despite protective strategy ventilation, and venovenous ECMO was administered. After one week of resuscitation using ECMO, his oxygenation improved and the patient was discharged in good condition.

Key words: transfusion-related acute lung injury; extracorporeal membrane oxygenation

INTRODUCTION

Blood transfusion-related acute lung injury (TRALI) has been reported to be associated with blood products, and is commonly seen with the transfusion of whole blood, packed red blood cells (PRBCs), fresh-frozen plasma (FFP) and platelets.[1] TRALI is a potentially fatal complication of massive blood transfusions, and typically develop within 6-72 h.[2] The incidence of TRALI has been estimated to be 0.014% to 0.02% per unit transfused.[1] The treatment of patients with TRALI is generally supportive, and the mortality rate is 5-8 %.[1] Extracorporeal membrane oxygenation (ECMO) is a technique for providing life support for patients experiencing both pulmonary and cardiac failure by maintaining their level of oxygenation.[3] The criteria for treatment with ECMO in adults vary between centers.[4] Several reports have shown that ECMO improves survival rates in patients with acute respiratory distress syndrome (ARDS) and other types of severe pulmonary failure.[5-7] Here, we present a patient with severe TRALI that was refractory to standard therapy, yet who was successfully resuscitated using ECMO.

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CASE REPORT

A previously healthy 20-year-old man suffered from close fractures of the right clavicle and scapular neck and the left pubic ramis, and urethral disruption, after being crushed by a 300 Kg iron shelf. No focal lesions were seen in either lung parenchyma on the patient's chest X-ray. An emergency operation to check internal bleeding and establish continuity of the urethra was performed immediately after admission. The operation went smoothly and the patient tolerated it well. Transfusion with 6 units of PRBCs and 4 units of whole blood during operation. The endotracheal tube was removed the next day. The patient received blood product transfusion because his hemoglobin level continued to fall (6.9 g/dL). Fever, dyspnea and desaturation were observed 3 h after transfusion of whole blood (4 units) and fresh-frozen plasma (FFP; 4 units). Arterial blood gas (ABG) analysis showed an oxygen partial pressure (PaO_2) of 207.9 mmHg and the ratio of PaO_2 to fraction of inspired oxygen (FiO_2) was 207.9. At that time, the chest radiography showed infiltrations

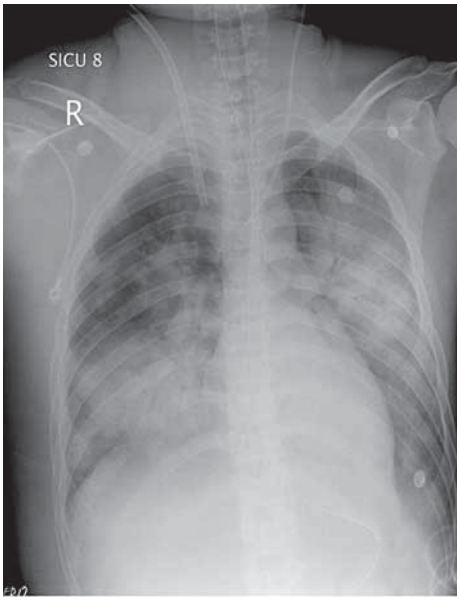

Fig. 1 Chest radiograph showing diffuse infiltrations of both lung zones

into both lung fields. The patient's central venous pressure (CVP) was 7 mmHg. However, he received blood product transfusions continuously because of the low hemoglobin (8.0 g/dL) and low platelet count (45000/μL), and more whole blood (4 units) and platelets (12 units) were transfused. Unfortunately, he developed severe dyspnea with oxygenation desaturation, tachycardia and low blood pressure 3 h after these transfusions. His vital signs revealed a blood pressure of 88/48 mmHg, a pulse rate of 118/min, a respiratory rate of 30/min and a body temperature of 39 °C. The patient received endotracheal tube intubation with mechanical ventilation. The ABG analysis showed a PaO_2 of 55.3 mmHg under mechanical ventilation with 100% oxygen. Chest radiography still showed infiltrations into both lung fields. An echocardiogram showed normal cardiac function and the patient's central venous pressure (CVP) was 11 mmHg. The laboratory data of blood and urine were within normal limits and sputum cultures showed negative findings. The clinical picture strongly indicated severe TRALI. The fulminant symptom was similar to ARDS. Thus, mechanical ventilation with a lung protective strategy was performed. He was ventilated under a pressure control mode, with a tidal volume of 6 mL/kg to keep the plateau pressure below 30 cmH_2O at a rate of 20 breaths per minute.

Unfortunately, his gas exchange deteriorated further during 16 h of mechanical ventilation, with a ratio of PaO_2 to fraction of inspired oxygen (FiO_2) of 41.4.

Table 1 Clinical characteristics and laboratory parameters of the patient with TRALI before on ECMO

Characteristics	Day of admission			
	Day 1	Day 2	Day 3	Day 4
Body temperature(°C)	34.6	38	39	36.8
CVP level (mmHg)	17	1	7	14
Lab Data				
WBC(/μl)	12540	7020	5310	4190
Hb(g/dl)	11	11	6.9	7.3
HCT(%)	24.6		20.3	20.6
PLT(/μl)	191000		45000	81000
Arterial blood gas				
pH			7.381	7.435
PaO_2/FiO_2 ratio			207.9	41.4
02SAT(%)			99.4	78.5
I/O	-3205	+3165	+1956	
Blood transfusion				
WB(U)	4		8	
PRBC(U)	6			
FFP(U)			4	
PLT(U)			12	
CxR finding	normal		Diffuse infiltration	Diffuse infiltration

Key: CVP, central venous pressure; Lab, laboratory; WBC, white cell count; Hb, haemoglobin; HCT, Hematocrit; PLT, Platelete; 02SAT, O2 saturation; I/O, input/output; WB, whole blood; PRBC, packed red cell; FFP, Fresh-frozen plasma

Therefore, ECMO was suggested. At this point, his chest radiography still showed diffuse infiltrations over both lung fields (Fig. 1). A venovenous ECMO, using a 21-Fr cannula in the right femoral vein and a 17-Fr cannula in the right internal jugular vein, was performed. The initial ECMO blood flow was 3.0 L/min and the initial sweep gas flow was 6 L/min. Mechanical ventilation with a lung-protective strategy was continued during ECMO support. Intravenous hydrocortisone (100 mg every 8 h per day) was administered for 2 days. Seven days after ECMO support, his pulmonary infiltration had resolved with improved gas exchange and he was weaned off ECMO. Thirteen days after withdrawing the ECMO support, he received an open reduction and internal fixation of his right scapula and right clavicle. Thirty-eight days after admission, he was discharged from our hospital in good condition.

DISCUSSION

The patients' clinical characteristics are shown in Table 1. TRALI is a well-recognized complication of blood transfusion that is characterized by dyspnea, hypotension, and hypoxemia that usually develops during or within 6-72 h after transfusion.[2] In our patient, he also developed TRALI within 72h after massive transfusion. The differential diagnosis of TRALI in our case includes pneumonia with ARDS, other causes of pulmonary edema, such as volume overload, congestive heart failure, and fat embolism syndrome. The sputum and blood cultures showed negative finding, so pneumonia with ARDS can be excluded. Besides, Pulmonary edema and congestive heart failure are less likely due to low central venous pressure and normal echocardiogram. Fat embolism syndrome (FES) is a rare complication occurring in 0.5 to 2% of patients following a long bone fracture.[8] Our patient has no long bone fracture and doesn't conform to the criteria of FES by Gurd and Wilson.[9] And the chest X-ray of FES should reveal "snow storm appearance," caused by diffuse bilateral alveolar infiltration that is not consisted with our patient.[9] Moreover, fat embolism syndrome remains a diagnosis of exclusion and is based on clinical criteria. Therefore, TRALI is diagnosed.

The risk of TRALI is higher with transfusion of plasma-rich blood products, FFP and platelets than with PRBCs.[10] A higher mortality for patients with TRALI has been reported following transfusion with FFP.[11] However, a recent study shows that male-predominant plasma strategy can reduce the risk of TRALI effectively.[12] Clinicians should practice this strategy to prevent TRALI or to transfuse blood products to the patient with suspected TRALI due to clinical conditions.

TRALI can be a self-limiting condition, so the treatment is the same as ARDS of any cause.[1,11] Steroids are usually administered, although there is little evidence to support their use, and diuretitcs may worsen the outcome secondary to intravascular volume depletion.[13] In this patient, despite our lung-protecting strategy of mechanical ventilation, severe hypoxemia with a PaO_2/FiO_2 ratio of 41.4 was observed. This indicated a high risk of death, so ECMO assistance therapy was warranted.[14]

ECMO has also been reported to benefit newborns with acute respiratory failure.[4] In adult patients with ARDS, two large randomized controlled trials failed to show an advantage of ECMO over conventional treatment.[3] However, such patients treated with ECMO have shown good survival rates—in excess of 50%—in uncontrolled studies in European centers.[4] Furthermore, in one prospective, randomized trial of 180 patients comparing ECMO with conventional ventilation, ECMO showed a lower incidence of mortality or severe disability than that with conventional ventilation.[3]

Most patients of TRALI recover within 72 hours and treatment is supportive, rarely requiring more than mechanical ventilation.[1] Resuscitation using ECMO for severe TRALI have been reported rarely. To our knowledge, three life-threatening cases of TRALI successfully treated with ECMO have been reported.[15,16,17]

Our case differs from previous cases in that ECMO was used to manage more severe and life-threatening hypoxemia due to PaO_2/FiO_2 ratio of 41.4, despite receiving mechanical ventilation with lung protective strategy. Use of ECMO for TRALI-induced lethal hypoxemia enables us to reduce the patient's FiO_2 and peak inspiratory pressure, moreover, prevent ventilator-induced lung injury.[15]

In conclusion, although ECMO is not yet a standard therapeutic modality for patients with TRALI, we suggest that ECMO may be appropriate therapy for severe cases who do not respond to supportive treatment on the basis of the outcome of our report and previous cases,.

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