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Case report

Severe transfusion-related acute lung injury managed with extracorporeal membrane oxygenation (ECMO) in an obstetric patient

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Abstract Transfusion-related acute lung injury (TRALI) is the leading cause of transfusion-related mortality in the United States. Management is usually supportive, including supplemental oxygen, intravenous fluids, and mechanical ventilation if necessary. Most patients recover within 72 hours. We present a nearly fatal case of TRALI in an obstetric patient, which was successfully managed with extracorporeal membrane oxygenation (ECMO).
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1. Introduction

Transfusion-related acute lung injury (TRALI), defined by the NIH working group, is new acute lung injury (ALI) occurring during or within 6 hours of a transfusion [1,2]. Acute lung injury is acute hypoxemia with an arterial oxygen concentration/fraction of inspired oxygen (PaO_2/FIO_2) ratio ≤300 mmHg or oxygen saturation ≤90% when a patient is breathing room air, found together with the appearance of bilateral infiltrates in the absence of left atrial hypertension [1,2].

All plasma-containing blood products have been implicated in TRALI, including packed red blood cells (PRBCs), whole blood, intravenous (IV) gamma globulin, and cryoprecipitate but, most commonly, whole blood-derived platelet concentrates and fresh frozen plasma (FFP) [3]. Most patients recover within 72 hours and treatment is supportive, rarely requiring more than mechanical ventilation [3].

We present a case of of severe TRALI, which was nearly fatal in an obstetric patient, but was successfully managed with extracorporeal membrane oxygenation (ECMO).

2. Case report

A 22 year-old, gravida 3, para 2 woman presented at 40 weeks' gestation with mild vaginal bleeding. She had a history of one prior classical cesarean delivery and she was to undergo an urgent repeat cesarean delivery. Combined

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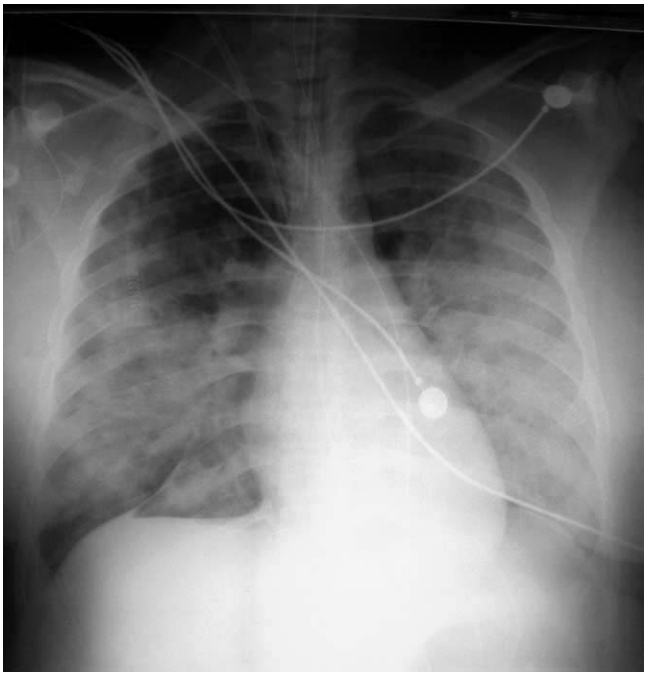

Fig. 1 Chest radiograph showing bilateral symmetrical alveolar opacities.

spinal-epidural (CSE) anesthesia was performed without complications using 1.2 mL of 0.75% hyperbaric bupivacaine, 20 μg of fentanyl, and 0.3 mg of preservative-free morphine intrathecally.

Following delivery of the infant, there was great difficulty in achieving hemostasis despite a well contracted uterus. There were dense adhesions between the myometrium, the anterior abdominal wall and urinary bladder, and two large myomas were present. Extension of the previous classical cesarean section scar occurred during delivery, which was difficult to repair. A great amount of tension was present during attempts at uterine closure, and atony occurred. Intramuscular methylergonovine and two doses of intramyometrial carboprost tromethamine were given. She remained hemodynamically stable for the next 45 minutes and anesthesia was maintained with 3% chloroprocaine via the epidural catheter. Lactated Ringer's solution was used for intravascular volume replacement.

Transfusion with PRBCs was started when the patient became hypotensive. Fresh-frozen plasma and platelets were also transfused to treat significant oozing in the surgical field. A supracervical hysterectomy was ultimately performed and hemostasis was achieved. Estimated total blood loss was 4 L. A total of 6 L of Ringer's lactate, 500 mL of hetastarch, 7 units of PRBCs, 6 units of FFP, and 10 units of platelets was administered.

During skin closure, the patient's oxygen saturation began falling despite supplemental oxygen. Her breathing became labored and bilateral crackles were heard. Furosemide 20 mg intravenously was given. The patient developed mental status changes and the oxygen saturation decreased to 83%. The patient's trachea was then intubated and mechanical ventilation was started with 100% O_2. Copious amounts of frothy sputum were noted in the endotracheal tube. The furosemide dose was repeated.

The patient was transferred to the surgical intensive care unit (SICU) where initial chest radiography showed bilateral symmetrical alveolar opacities (Fig. 1). A pulmonary artery (PA) catheter was placed and showed a pulmonary capillary wedge pressure of 16 mmHg, PA pressure of 46/30 mmHg, and a cardiac output (CO) of 6.0 L/min.

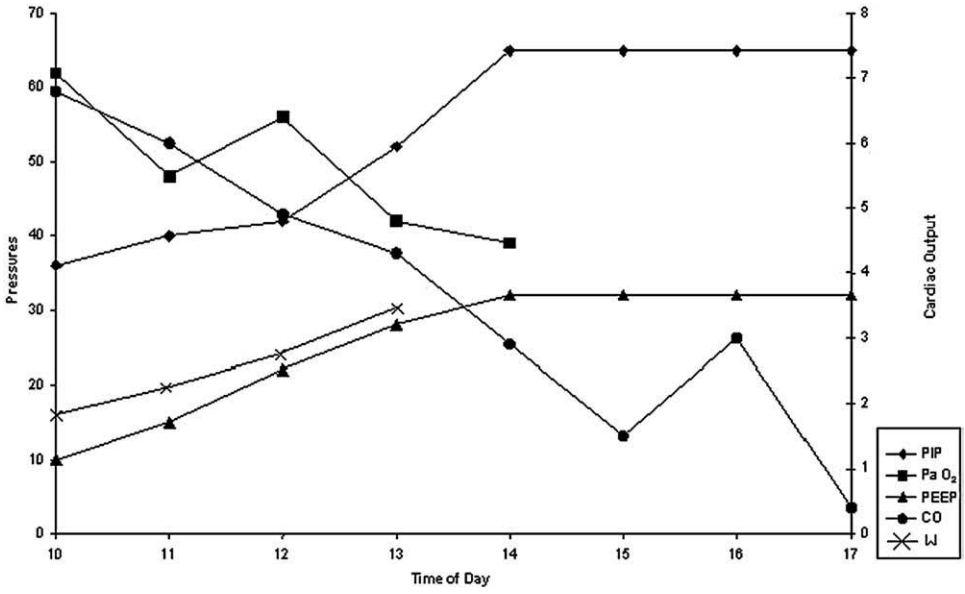

Fig. 2 Falling cardiac output (CO, L/min), worsening hypoxemia, and progressively higher pulmonary capillary wedge pressure over time associated with increasing ventilator support. PIP = peak inspiratory pressure (cm H_2O), PEEP = positive end-expiratory pressure (cm H_2O), PaO_2 = partial pressure of oxygen (mmHg), and W = pulmonary capillary wedge pressure (mmHg).