**Transfusion-related Acute Lung Injury During Liver Transplant: Case Report**

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**Introduction**

Transfusion-related acute lung injury (TRALI) is defined as noncardiogenic pulmonary edema temporally related to the transfusion of blood products. Brittingham reported the first link between symptoms of ALI, transfusion and onset of pulmonary edema temporally related to the transfusion of blood products. We present a patient who, while undergoing orthotopic liver transplantation, developed acute pulmonary edema within minutes of administration of fresh frozen plasma (FFP).

**Intraoperative Course**

A 71-year-old female presented for orthotopic liver transplantation. She was brought to the operating room and underwent uneventful induction of general anesthesia, vascular access placement, and tracheal intubation. The operative field was noted to have continued bleeding and blood products were administered to improve hemostasis. Within minutes of administration of FFP, copious amounts of pale yellow, frothy fluid filled the endotracheal tube and the patient’s oxygen saturation dropped from 100% to 90%. The patient was placed on 100% FiO2, intermittent suctioning removed 1 L of fluid, and IV furosemide was given. Hemodynamics and oxygenation stabilized, the operation was completed without further difficulty, including transfusion of additional blood products.

**SICU Course**

The patient arrived in the SICU on 100% FiO2 and 10 of PEEP. Vasopressor and norepinephrine were being administered due to persistent hypotension, likely secondary to large intravascular volume shifts during the procedure (approximately 7 liters of ascites were drained from her abdomen). Over the next 36 hours, the goal of therapy shifted from volume resuscitation to diuresis and weaning from mechanical ventilation. By the end of post-op day #2 the patient was on 40% FiO2 and spontaneously ventilating with CPAP. Prolonged encephalopathy prevented successful extubation and the patient had a tracheostomy placed post-op day #6. She was successfully liberated from mechanical ventilation the next day. The remainder of her hospital course was uneventful and she was discharged to rehab three weeks later. She has had multiple type and screens drawn and her antibody screen remains persistently negative.

**TRALI: Differential Diagnosis**

The differential diagnosis of TRALI includes transfusion associated circulatory overload (TACO), left ventricular failure, exacerbation or progression of ALI from another cause, ARDS, sepsis, trauma, smoke inhalation, aspiration pneumonitis or pneumonia. The patient had no prior ALI, no active infections, and had not been subject to trauma. Intraoperative TEE demonstrated normal function of the left and right ventricles. TACO was ruled out on the basis of preoperative hypotension, removal of 7 L of ascites, a normal TEE, no changes on EKG, and a temporal relationship with the transfusion of 2 units of FFP.

<table>
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<th>ABG</th>
<th>Post-Induction</th>
<th>Onset of TRALI</th>
<th>Post-op</th>
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<tr>
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<td>100%</td>
<td>60%</td>
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<tr>
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<tr>
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<tr>
<td>O2 sat%</td>
<td>99</td>
<td>88</td>
<td>100</td>
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</tbody>
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**TRALI: Figure 2. Imaging**

- **At left:** Pre-op CXR demonstrating mild left lower lobe collapse, but otherwise clear lungs.
- **At right:** CXR immediately post-op, showing diffuse bilateral infiltrates consistent with pulmonary edema.

**TRALI: Definition and Current Concepts**

Brittingham reported the first link between symptoms of ALI, transfusion and leukoagglutinin in 1957. The term TRALI was coined by Poppas in 1985 after describing a series of cases of ALI in association with leukoagglutinin in the blood component. Partially because of vigilance in preventing the spread of viral illnesses, TRALI has, according to the FDA, become the leading cause of morbidity and mortality associated with blood transfusion. In 2005, the National Heart, Lung, and Blood Institute convened a panel to provide a clinically useful definition which is, “new ALI occurring during or within 6 hours after a transfusion, in patients with or without risk factors for ALI other than transfusion”. There are two theories as to how injury to the lung occurs in TRALI: The “Antibody Hypothesis” which invokes immune priming and the introduction of a TRALI producing agent. Priming sensitizes the vascular endothelium to a trigger which may be the same agent at a higher dose or another agent entirely. The trigger in TRALI appears to be lipids from stored blood. The “Antibody Hypothesis” focuses on donor derived anti-leukocyte antibodies that trigger increased vascular permeability in the lung. HLA class I and II from female, multiparous donors have been implicated in cases of TRALI. The components with the highest risk of producing TRALI are FFP and platelets. Treatment is supportive, with sequelae resolving within 96 hours of onset. The incidence of TRALI in liver transplant patients is 1.3% and was found to be associated with plasma-containing products only (FFP, platelets). Rate of infection was directly related to PRBC administration in a dose dependent manner. Given the complications related to transfusion, it is prudent to minimize transfusion of blood products as much as possible.

**References**


**Abbreviations**

- ABG: arterial blood gas
- TEE: transesophageal echocardiogram
- TACO: transfusion-associated circulatory overload
- TRALI: Transfusion Related Acute Lung Injury