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Nathanael Slater
University of Massachusetts Medical School

Nicholas C. Watson
University of Massachusetts Medical School

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

Nathanael A. Slater D.O., Nicholas C. Watson M.D.
Department of Anesthesia, University of Massachusetts Medical School, Worcester MA

Introduction

Transfusion-related acute lung injury (TRALI) is defined as noncardiogenic pulmonary edema temporally related to the transfusion of blood products.1 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 an 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, foamy fluid filled the endotracheal tube and the patient's oxygen saturation dropped from 100% to 80%. The patient was placed on 100% FiO2 intermittent suctioning removing 1L 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. Vasopressin 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 weaned from mechanical ventilation the next day. The remainder of her hospital course was uneventful and she was discharged to rehab three weeks later. As of this past January, on follow-up in transplant clinic, she is doing well, has a healthy appetite, and is able to participate in the day to day activities of her family life. She has had multiple trips and someone down 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 pneumonia 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 hypovolemia, 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.

TRALI: Figure 1. Intraoperative Diagnosis

At left: One of two containers of pulmonary edema suctioned from the patient's ETT.

At right: Sequential ABGs obtained intraoperatively and immediately postop. Induction was at 5:45 AM, transfusion of FFP occurred at 6:45 AM. FiO2 at induction was 40% with 5 of PEEP on volume cycled ventilation. The second ABG is at 06:51 at which time copious amounts of fluid were being removed from the ETT via suction. FiO2 was 100%, PEEP 10. Post-operatively, almost 12 hours later, ventilation, oxygenation, and respiratory mechanics had improved significantly. The patient was on 40% FiO2 with 5 of PEEP.

<table>
<thead>
<tr>
<th>ABG</th>
<th>Post-Induction</th>
<th>Onset of TRALI</th>
<th>Post-op</th>
</tr>
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<tbody>
<tr>
<td>FiO2</td>
<td>40%</td>
<td>100%</td>
<td>60%</td>
</tr>
<tr>
<td>pH</td>
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<tr>
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<td>16.2</td>
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</tr>
<tr>
<td>O2 sat%</td>
<td>99</td>
<td>98</td>
<td>100</td>
</tr>
</tbody>
</table>

TRALI: Figure 2 Imaging

At left: Pre-op 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 a temporal relationship with the transfusion of blood products.1,2 We present a patient who, while undergoing orthotopic liver transplantation, developed acute pulmonary edema within minutes of administration of fresh frozen plasma (FFP). The incidence of TRALI in liver transplant patients is 1.3% and was found to be associated with plasma-containing products only (FFP), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1 The components with the highest risk of producing TRALI are FFP and platelets.2 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), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1 The components with the highest risk of producing TRALI are FFP and platelets.2 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), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1 The components with the highest risk of producing TRALI are FFP and platelets.2 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), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1 The components with the highest risk of producing TRALI are FFP and platelets.2 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), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1 The components with the highest risk of producing TRALI are FFP and platelets.2 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), and not platelets.1 The components with the highest risk of producing TRALI are stored blood and prestorage leukoreduction.3 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.1

References

4. Benson AB, Burton AR. Differential effects of plasma and red blood cell transfusions on acute lung injury and infection risk following liver transplantation. Traspl Int 2008 21:258

Abbreviations

ABG arterial blood gas analysis
ALI acute lung injury
ARDS acute respiratory distress syndrome
CPP cerebral perfusion pressure
CXR chest x-ray
ETT endotracheal tube
FiO2 fraction of inspired oxygen
PEEP positive end expiratory pressure
SICU surgical ICU
TACO transfusion associated circulatory overload
TRALI transfusion associated acute lung injury
TGA transesophageal echocardiogram
TEE transthoracic echocardiogram
TRIPS transesophageal regional ischaemia perfusion study
TROCS transesophageal regional oxygenation and cardiac output study
TTP time to peak
TRPL transient right to left shunt
WBC white blood cell
WMT worst measurable time
XAT xenon absorption test
YAB xenon absorption breath test