Transfusion-Related Acute Lung Injury during Liver Transplant: Case Report

Nathanael Slater

University of Massachusetts Medical School

Let us know how access to this document benefits you.
Follow this and additional works at: https://escholarship.umassmed.edu/anesthesiology_pubs

Repository Citation

This material is brought to you by eScholarship@UMassChan. It has been accepted for inclusion in Anesthesiology and Perioperative Medicine Publications by an authorized administrator of eScholarship@UMassChan. For more information, please contact Lisa.Palmer@umassmed.edu.
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. Initial reports of TRALI described cases occurring over 6-8 hours post-transfusion; however, recent reports have described TRALI occurring within minutes of transfusion. Brittingham reported the first link between symptoms of acute lung injury (ALI), transfusion and leukocyte antibodies in 1957.1,2 This was followed by Popovsky in 1983 who reported the first case of TRALI.3 The term TRALI was coined by Popovsky in 1983 and is defined by the FDA as acute lung injury temporally related to the transfusion of fresh frozen plasma.4 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 admitted to the operating room over night with a right upper quadrant abdominal mass. Preoperative laboratory values were notable for anemia (Hgb 8.9 g/dL) and ferritin levels of 6730 ng/mL. She was on nonsteroidal anti-inflammatory medications for chronic arthritis and had a history of alcohol abuse. Physical exam was notable for nondescript liver mass, normal bowel sounds, and no ascites. On admission, her orders included a liquid diet, no alcohol, and furosemide. Labs were sent for a complete blood count, liver function tests, and coagulation profile. The patient arrived in the operating room 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 stabilized and hemodynamically optimized.

Intraoperative TRALI diagnosis.

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

At right: Sequential ABGs obtained intraoperatively and immediately post-op. Induction was at 54/80 AM, with FiO2 at 40% and 5 of PEEP on volume cycled ventilation. The second ABG was at 06:51 with 100% FiO2, 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 65% FiO2 with 5 of PEEP.

TRALI: Figure 1, Intraoperative Diagnosis

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.

SIU Course

The patient arrived in the SICU on 100% FiO2 and 10 of PEEP. Vasopressin and norepinephrine were being administered due to persistent hypotension. She was noted to have continual bleeding and blood products were administered to improve hemostasis. Within minutes of administration of FFP, the patient required 2, intermittent suctioning, and 1L of fluid from the endotracheal tube. The patient had received 5 of PRBC, 5 of FFP, 10 of platelets, and 10 of cryoprecipitate. At induction, 40% FiO2 was maintained. At 06:51, FiO2 was 100%, PEEP 10. Within minutes of administration of FFP, the patient’s oxygen saturation dropped from 100% to 90%. 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.

TRALI: Figure 3, Imaging

At left: Post-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: 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, had 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 hemodynamics, 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: Definition and Current Concepts

Brittingham reported the first link between symptoms of ALI, transfusion and leukocyte antibodies in 1957.6 The term TRALI was coined by Popovsky in 1983 after describing a series of cases of ALI in association with leukoagglutinins in the blood component.7 Partially because of vigilance in preventing the spread of viral illness, TRALI has, according to the FDA, become the leading cause of transfusion-related mortality and morbidity 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 transfusional trauma.”8 There are two theories as to how injury to the lung occurs in TRALI. One is the “Two Hit Hypothesis” which involves immune priming and the introduction of a TRALI producing agent.9 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.3 The “Antibody Hypothesis” focuses on donor derived anti-leukocyte antibodies that trigger increased vascular permeability in the lung.10 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.6 Treatment is supportive, with sequelae resolving within 36 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).6 Rate of infection was directly related to PRBC administration in a dose dependent manner.4 Given the complications related to transfusion, it is prudent to minimize transfusion of blood products as much as possible.6

References


Abbreviations

ABG: arterial blood gas
ALI: acute lung injury
ARDS: acute respiratory distress syndrome
CXR: chest x-ray
EKG: electrocardiogram
ETT: endotracheal tube
FiO2: fraction of inspired oxygen
HCA: hospital care area
PRBC: packed red blood cells
TACO: transfusion-associated circulatory overload
TRALI: transfusion-related acute lung injury
TTE: transesophageal echocardiogram

Abbreviations

ABG: arterial blood gas
ALI: acute lung injury
ARDS: acute respiratory distress syndrome
CXR: chest x-ray
EKG: electrocardiogram
ETT: endotracheal tube
FiO2: fraction of inspired oxygen
HCA: hospital care area
PRBC: packed red blood cells
TACO: transfusion-associated circulatory overload
TRALI: transfusion-related acute lung injury
TTE: transesophageal echocardiogram