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ACUTE RESPIRATORY FAILURE DURING ROUTINE BLOOD TRANSFUSION: A CASE REPORT AND REVIEW OF THE LITERATURE

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☐ **Abstract—Background: Transfusion medicine is a common practice in the emergency department (ED) and other outpatient settings, and may be complicated by a low rate of potentially fatal transfusion-related reactions. Objectives: This article presents a case of transfusion-related acute lung injury (TRALI) diagnosed and treated in the ED and reviews the differential diagnosis of acute transfusion reactions. Case Report: A 74-year-old woman presented to the ED from the hospital's transfusion center with fever and respiratory distress immediately after the start of her second unit of red blood cell transfusion. Chest radiograph demonstrated a pattern consistent with acute respiratory distress syndrome (ARDS). After 48 h of respiratory support and antibiotic therapy, the patient's condition improved. Conclusion: TRALI is a clinical diagnosis with presentation similar to that of ARDS. Prompt differentiation from other transfusion reactions and initiation of appropriate treatment is crucial in minimizing the morbidity and mortality associated with this syndrome. Published by Elsevier Inc.**

☐ **Keywords—blood transfusion reaction; TRALI; acute lung injury**

INTRODUCTION

Approximately 30 million units of red blood cells (RBCs), platelets, and plasma are transfused in the United States (US) each year, many of these in the Emergency Department (ED) (1). Although transfusion-related adverse events remain relatively uncommon, more than 50 transfusion-related deaths are reported annually to the Food and Drug Administration (1). Prompt differentiation between the major life-threatening transfusion reactions and implementation of care appropriate to the etiology is important in decreasing morbidity and mortality. This article presents a case of transfusion-related acute lung injury (TRALI) developing during outpatient blood transfusion, diagnosed and treated in the ED. A summary of the differential diagnosis and appropriate treatment for this and other acute transfusion reactions is provided. Per our review, no previous case reports exist of TRALI diagnosed and treated in the ED.

CASE REPORT

A 74-year-old woman presented to the ED from the hospital's transfusion center with acute onset of respiratory distress developing within 10 min of starting the second of two units of a routine packed RBC transfusion. Past medical history included pancreatic cancer, chronic anemia, hypertension, diabetes mellitus, hypothyroidism, and biliary stent. Review of systems was positive for acute dyspnea and chest pressure. The patient and family endorsed recent mild malaise and subjective fever, but denied chills, sweats, dysuria, cough, or significant dyspnea prior to the transfusion.

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On examination, the patient was in obvious respiratory distress, communicating via one-word statements and head nodding. Vital signs showed a temperature of 38.9°C (102.1°F), heart rate of 151 beats/min, blood pressure 207/106 mm Hg, respiratory rate 44 breaths/min, and pulse oximetry of 70% on room air, improving to 98% on 15 L/min oxygen by face mask. Physical examination showed no jugular venous distension or any angioedema. Her pulmonary examination demonstrated rales and rhonchi. Her cardiac examination revealed a regular tachycardic rhythm without murmur or rub. Extremities showed moderate pulses with no edema, calf tenderness, or palpable cords. Skin was warm and dry without rash. A peripherally inserted central catheter (PICC) line was observed in the patient's right upper extremity without associated cellulitis or thrombosis.

Electrocardiogram was normal except for sinus tachycardia. Laboratory analysis revealed a serum glucose of 310 (mg/dL), potassium of 2.9 (mmol/L), total bilirubin 3.7 (mg/dL), alkaline phosphatase 271 (U/L), and serum glutamic-oxaloacetic transaminase 103 (U/L). Other parameters, including serum electrolytes, creatinine, lactic acid, liver function, prothrombin time/partial thromboplastin time, and complete blood count, were within normal limits. Bilateral infiltrates were noted on chest radiograph (Figure 1).

Initial ED treatment included a trial of bilevel positive airway pressure followed by rapid sequence intubation and mechanical ventilation. During laryngoscopy, copious frothy secretions were noted. After obtaining blood cultures, piperacillin/tazobactam and vancomycin were started, as well as gentle hydration. A presumptive diagnosis of TRALI was made prior to intensive care unit (ICU) transfer.

ICU testing included cardiac echocardiography on the day of admission showing an ejection fraction of 35%, normal pulmonary artery pressure, and no vegetations. Abdominal and pelvic computed tomography showed no foci of infection. ED blood cultures grew 4/4 bottles of *Enterobacter Cloacae* sensitive to piperacillin/tazobactam. Repeat blood cultures remained positive despite continued piperacillin/tazobactam therapy, leading to the removal of the patient's PICC line as a likely source of continued bacteremia. The patient's respiratory status improved over the ensuing 48 h with conservative management and gentle intravenous hydration. The patient was successfully extubated on hospital day 3, and was discharged home at her baseline functional status on hospital day 7.

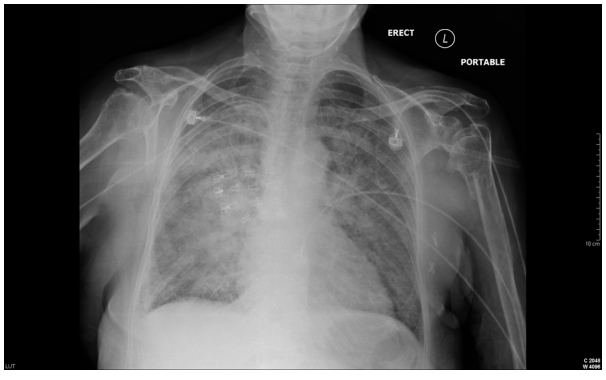

Figure 1. Chest radiograph upon Emergency Department arrival demonstrating bilateral infiltrates.

DISCUSSION

TRALI complicates transfusions in one of every 5000 units of packed RBCs, one in 2000 units of plasma-containing components, and one of every 432 units of whole blood (2). Significantly higher percentages, up to 8% incidence, have been found in critically ill patients receiving transfusion (3–5). Despite the low overall incidence, it is associated with a significant overall mortality of 5–10%, and is the leading cause (approximately 50%) of transfusion-associated deaths in the US (1,6). Current animal models on the pathophysiology of this syndrome suggest that transfusion with leukocyte-containing blood products in patients with an "immune priming step" such as recent surgery, bacteremia, or other physical stress produces an acute neutrophilic response. This immune response interacts with platelets in the lung microcirculation, resulting in neutrophil sequestration, endothelial damage, and massive capillary leak in the pulmonary vasculature (7). We believe TRALI to have been triggered in our patient due to low-grade bacteremia related to her PICC line.

The diagnosis of TRALI is clinical, with patients presenting with signs and symptoms of acute lung injury or acute respiratory distress syndrome. Consensus definition by the National Heart, Lung, and Blood Institute defines the syndrome as severe hypoxemia (O_2 saturation < 90% on room air) and bilateral pulmonary infiltrates presenting within 6 h of transfusion initiation, accompanied by lack of evidence of pulmonary artery hypertension or generalized fluid overload (2). Fever, blood pressure changes, and tachycardia are common. Whereas our patient presented with systemic hypertension, patients also commonly present with hypotension (8). Differential diagnosis for TRALI includes transfusion-associated circulatory overload (TACO), anaphylaxis, sepsis, and acute hemolytic transfusion reaction.

Similar to TRALI, TACO presents with hypoxemia, respiratory distress, hypertension, and pulmonary edema on radiography (4). However, it is unlikely to produce high fever. In addition, distended neck veins and other markers of generalized volume overload are hallmarks