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Case Report

The unforeseen complication of blood transfusion: Transfusion-related acute lung injury – A case report

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ABSTRACT

Transfusion-related acute lung injury (TRALI), a rare yet potentially fatal complication, manifests as acute-onset non-cardiogenic pulmonary edema accompanied by severe hypoxemia. In this report, we present the case of a 25-year-old female who experienced TRALI shortly after undergoing a blood transfusion and exhibited a favorable response to steroid therapy.

Keywords: Transfusion-related acute lung injury, Respiratory distress, Systemic steroid therapy, Oxygen therapy

INTRODUCTION

Transfusion-related acute lung injury (TRALI) is a rare but serious syndrome which presents within the first 6 h of transfusion of any blood products as non-cardiogenic pulmonary edema with hypoxemia.[1,2] According to reports, TRALI has an estimated incidence of approximately 1 in 5000 transfusions. While any blood product has the potential to lead to TRALI, the risk of its onset is heightened when blood products containing more than 50 mL of plasma are used or when platelet suspensions stored in the blood bank for a prolonged period are transfused.[3] TRALI is a prominent cause of mortality associated with transfusion, and thus, it is crucial to maintain close and frequent monitoring of patients, especially in vulnerable groups such as patients with sepsis, those on mechanical ventilation, and those undergoing cardiovascular surgery or having end-stage liver disease.[4]

CASE REPORT

A 25-year-old lady with no significant medical history, post lower segment cesarean section (LSCS) day 1, was transferred to the emergency department from the obstetrics and gynecology department after complaint of acute onset of breathlessness for 3 h following transfusion of one unit of packed red blood cells 6 h back from a female donor. At presentation, the patient was having tachycardia (pulse rate = 124/min), blood pressure = 82/56 mm Hg, tachypnea (respiratory rate = 30/min), and oxygen desaturation (SpO_2 = 76% on room air). Respiratory sounds were diminished bilateral and rales were heard bilaterally all over the chest. The patient was immediately provided 8–10 L/h oxygen therapy following which the patient maintained SpO_2 of 95% and no mechanical ventilation was required.

Blood tests revealed hemoglobin: 9.1 g/dL, packed cell volume: 27.8%, white blood cells: 9,900/mm^3, platelet count: 1.2 lakh/mm^3, random blood sugar: 91 mg%, blood urea: 38 mg/dL, serum creatinine: 1.1 mg/dL, total serum bilirubin: 1.1 mg/dL, indirect bilirubin: 0.8 mg/dL, alanine aminotransferase: 38 IU/L, aspartate aminotransferase: 52 IU/L, total serum protein: 5.6 g/dL, serum albumin: 3.4 g/dL, serum Na^+: 131 meq/L, serum K^+: 4.3 meq/L, and serum Ca^{2+}: 9 mg/dL.

Chest X-ray posteroanterior (PA) view revealed bilateral diffuse irregular reticulonodular infiltrates [Figure 1]. Electrocardiogram of the patient revealed no abnormality. The patient was managed on the line of TRALI with an injection methylprednisolone 10 mg/kg/day for 3 days, along with an oxygen supplement. Gradually, the respiratory distress of the patient regressed and oxygen requirement decreased and the patient was weaned off of oxygen after 3 days following which the patient was stable at room air. Repeat chest X-ray PA view was done after 5 days which showed regression of infiltrates [Figure 2].

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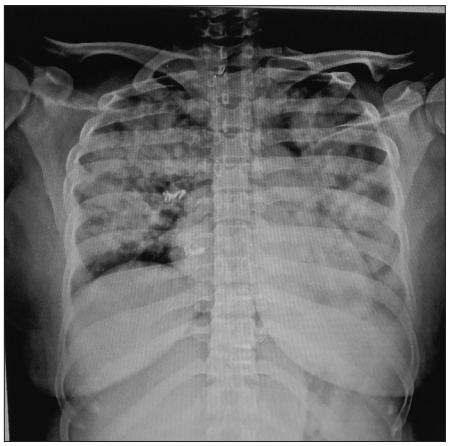

Figure 1: Chest X-ray posteroanterior (PA) view showing bilateral diffuse irregular reticulonodular infiltrates.

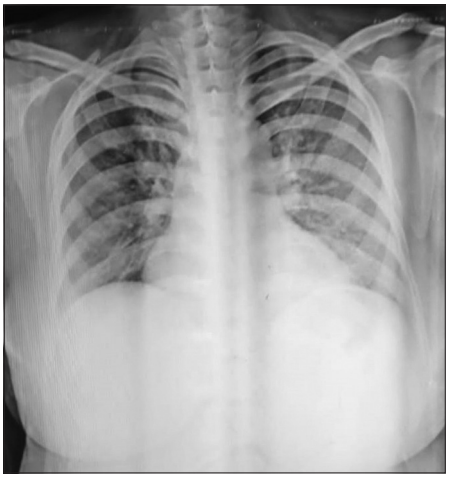

Figure 2: Chest X-ray posteroanterior (PA) view showing regression of infiltrates.

DISCUSSION

Back in 1983, Popovsky *et al*. reported the first instance of TRALI when five patients experienced non-cardiogenic pulmonary edema symptoms within 4 h of blood product transfusions.[5]

TRALI risk factors encompass patients with low interleukin-10 levels, systemic inflammation, shock, high peak airway pressure during mechanical ventilation, malignancies, and individuals undergoing cardiovascular and liver surgery.[6]

The exact pathophysiology of TRALI is not yet fully elucidated. However, it is hypothesized that the recipient of blood products experiences an inflammatory response that sensitizes the neutrophils within the lungs. Activation of these neutrophils by alloantibodies from the donor leads to an amplified local inflammatory process, causing damage to lung tissue and pulmonary blood vessels, and ultimately leading to the development of non-cardiogenic pulmonary edema.[7]

The incidence of TRALI has been reported to be higher in cases where plasma is obtained from female donors. This is due to the presence of multiple human leukocyte antigen antibodies observed in parous female donors.[8]

TRALI also needs to be differentiated from allergic and anaphylactic reactions, transfusion-associated circulatory overload (TACO), sepsis, cardiogenic pulmonary edema, and transfusion-associated dyspnea all of which present with hypoxia following transfusion. TACO is also a significant contributor of mortality following blood transfusion and may be confused with TRALI due to their overlapping symptomatology.[9] In our case, the absence of elevated jugular venous pressure, gallop rhythm, murmur, and S3 sound was consistent with TRALI.

In the absence of targeted therapy for TRALI, symptomatic and supportive treatment forms the cornerstone of patient management. Immediate cessation of transfusion is essential for TRALI diagnosis. Providing oxygen therapy and stabilizing the patient's hemodynamics are crucial forms of support. Mechanical ventilation might be required for patients experiencing ongoing hypoxia despite receiving oxygen therapy. The role of steroids in TRALI management is still not clearly defined.[10] However, in our case, the patient showed a dramatic response to steroid therapy.

Enhancements in blood storage conditions, meticulous donor screening, vigilant bedside monitoring of transfusions, and recognition of high-risk factors before transfusion and thorough documentation of adverse events are all pivotal in diminishing transfusion-related morbidity and mortality.

CONCLUSION

The case report discussed above underscores the importance of recognizing predisposing risk factors for TRALI before the transfusion of blood products. In our case, female donor and major surgery (LSCS) could be the potential risk factors for developing TRALI. While the role of steroids in TRALI management is not well understood, yet, in our case, the patient exhibited a favorable response to systemic steroid therapy. In addition, healthcare professionals should be vigilant for TRALI in cases of acute respiratory distress that arise during and immediately after blood product transfusion.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The author(s) confirms that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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