CASE REPORT

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Transfusion-Related Acute Lung Injury: Trali

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ABSTRACT

Transfusion-Related Acute Lung Injury (TRALI) is a severe, life-threatening transfusion reaction characterised by the acute onset of non-cardiogenic pulmonary edema and hypoxemia occurring within six hours of blood product administration. A 55-year-old female with chronic iron deficiency anemia (Hb 7.1 g/dL) presented with fatigue, dyspnea, and melena, receiving one unit of packed red blood cells (PRBCs) while hemodynamically stable. After forty-five minutes of transfusion, she developed acute respiratory distress (SpO2 85%, RR 32, BP 90/55, HR 115, fever 38.5°C), bilateral crackles without Jugular Venous Distention (JVD) or S3, confirming TRALI over Transfusion associated circulatory overload (TACO). Transfusion was stopped; immediate management included BiPAP, high-flow oxygen, crystalloid bolus, norepinephrine, and diuretic avoidance. The patient showed significant recovery within 24 hours and was discharged on day 5 with no residual respiratory deficits and GI follow-up.

KEYWORDS

• Transfusion reaction • Non-cardiogenic pulmonary edema • Acute Lung Injury • Diuretic • Packed Red Blood Cells • Hypoxemia

INTRODUCTION

TRALI is a rare but serious complication of blood transfusion, characterised by the sudden onset of acute respiratory distress within six hours of receiving blood products. It had clinical features of acute onset of non-cardiogenic pulmonary oedema (lung fluid accumulation not caused by heart failure) and severe hypoxemia, hypotension and fever. TRALI is recognised as the leading cause of

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transfusion-related mortality. It often results from an immune or non-immune reaction where a pre-existing inflammatory state in the patient is followed by an activating substance in the donor blood, leading to massive neutrophil activation and damage to the lung capillaries that causes fluid to accumulate in the lungs. The condition is most commonly associated with plasma-containing blood products and can be life-threatening if not promptly recognized and managed.[1]

TRALI occurs through a "Two-Hit" mechanism where a recipient's pre-existing inflammatory state, such as sepsis or trauma (the First Hit), causes neutrophils to become "primed" and sequestered within the lung capillaries. The Second Hit involves the transfusion of activating substances either donor Human leukocyte antigen or Human neutrophil antigen (Anti-HLA/HNA) antibodies in Immune TRALI or biologically active lipids in Non-Immune TRALI, which trigger these sequestered neutrophils to release proteases and reactive oxygen species. This activation severely damages the endothelial lining and alveolar-capillary membrane, leading to a massive increase in capillary permeability and the rapid leakage of protein-rich fluid into the alveolar spaces. The resulting non-cardiogenic pulmonary edema causes acute hypoxemic respiratory failure and presents clinically as a form of Acute Respiratory Distress Syndrome (ARDS).[2]

BACKGROUND & EPIDEMOLOGY

Worldwide, TRALI is the leading cause of transfusion-related death with an estimated incidence of 1 in 5,000 plasma-containing transfusions, though rates may be higher in certain populations such as those in intensive care units. It is more common in patients with underlying conditions like sepsis, trauma, or recent surgery, and its incidence has declined due to improved screening and mitigation strategies. Mortality rates associated with TRALI range from 5% to 25%.[3]

The history of TRALI began with sporadic reports of acute respiratory distress following transfusion in the 1950s, though it wasn't formally recognised until 1985 by Dr Popovsky and Moore, who established the key diagnostic criteria of acute, non-cardiogenic pulmonary edema related to transfusion. In the 1990s, research identified the primary cause as the presence of Anti-HLA and Anti-HNA antibodies in donor plasma. In the early 2000s TRALI was assigned as the leading cause of transfusion-related fatality, prompting a major shift in blood bank practices.[4]

CASE REPORT

History

A 55-year-old female presented to the emergency department with a one-week history of progressive fatigue, dyspnea, lightheadedness, weakness, and intermittent melena. She had a past medical history significant for chronic iron deficiency anemia, for which she had received intermittent oral iron supplementation in the past, but compliance was poor. Further, she denied any history of peptic ulcer disease, liver disease, or recent surgery. There was no family history of bleeding disorders or malignancy.

On examination, she appeared pale and fatigued. Her vital signs on admission were: HR 92 beats/mts, BP 130/80 mm/Hg, RR18 breaths /mts and O_2 Saturation 97% on room air. Physical examination revealed pallor, mild abdominal tenderness in the epigastric region, and no signs of hepatosplenomegaly or peripheral stigmata of chronic liver disease. Initial laboratory investigations revealed haemoglobin of 7.1 g/dL, mean corpuscular volume (MCV) of 72 fL, serum ferritin of 8 ng/mL, and total iron-binding capacity (TIBC) of 450 µg/dL, confirming iron deficiency anemia.

Given her symptomatic anemia, a transfusion of two units of packed red blood cells (PRBCs) was suggested to improve oxygen-carrying capacity and prevent further complications. The transfusion was initiated while she was hemodynamically stable, with the intention to correct her anemia and support recovery.

The first unit of PRBCs was initiated under close monitoring. For the first 30 minutes, the patient remained stable. Approximately 45 minutes into the transfusion, the clinical picture changed abruptly.

Assessment

The patient began to experience acute, severe dyspnea, developed a persistent hacking cough and a profound sense of anxiety. On assessment, her vitals showed severe hypoxemia (O_2Sat 85% on Room Air) and Tachypnea (RR 32 breaths /minutes), coupled with hypotension

(BP 90/55 mmHg), tachycardia (HR 115 beats / minutes, and a fever (38.5 °C), indicating acute respiratory distress and systemic instability. The pulmonary finding of bilateral coarse crackles and decreased breath sounds confirms the presence of non-cardiogenic pulmonary edema (fluid-filled lungs). Crucially, the cardiovascular assessment showing no JVD and a lack of S_3 gallop is a key differentiating factor as it rules out hydrostatic/cardiogenic causes of pulmonary edema, such as TACO, strengthening the diagnosis of TRALI.

Lab Findings

Following the immediate cessation of the transfusion and notification of the blood bank, the diagnosis of TRALI was established based on the temporal relationship and key clinical and laboratory findings. The Arterial Blood Gas (ABG) confirmed acute lung injury with a PaO_2/ FiO_2 ratio of <300 mmHg. Imaging via Chest X-ray revealed new, bilateral, diffuse pulmonary infiltrates (pulmonary edema) alongside a normal cardiac silhouette. The elevation in neutrophil count reflects the underlying systemic inflammation. Crucially, the non- cardiogenic nature of the injury was strongly supported by the patient's normal B-type Natriuretic Peptide (BNP) 85 pg/ml, lack of JVD on physical exam, and normal cardiac enzymes, which effectively ruled out cardiac failure and TACO.

MANAGEMENT & POST-DISCHARGE FOLLOW-UP

The management of TRALI is focused on prompt recognition, cessation of the transfusion, and aggressive supportive care to address respiratory and hemodynamic instability. Immediate interventions included are, stopping the transfusion, providing high-flow oxygen and non-invasive ventilation (BiPAP) to improve oxygenation, and administering a small crystalloid bolus with vasopressor support (norepinephrine) to maintain perfusion in the setting of hypotension. A crucial therapeutic omission was the intentional avoidance of diuretics, as they could worsen the underlying hypovolemia and shock associated with this permeability-based injury.

As the patient's condition improved, supportive therapies were gradually weaned, transitioning from BiPAP to high-flow nasal cannula and eventually room air. Close monitoring in the intensive care unit ensured the timely detection of any deterioration and allowed for rapid intervention. Further, with appropriate supportive management and avoidance of harmful interventions, most patients with TRALI recover fully without residual respiratory or hemodynamic deficits, as explained in Table 1.

Table 1: Clinical Progression and Response to Treatment Timeline

Stage	Clinical Parameters and Monitoring Outcomes
1-2 hours (Immediate Post Intervention)	**Vital signs:** HR 110 bpm, BP 95/60 mmHg (on norepi), RR 28, SpO2 92% on NRB **Respiratory status:** Persistent crackles, improved work of breathing **Hemodynamic status:** Stabilizing on norepinephrine 5 mcg/min + 250 mL crystalloid **Clinical notes:** BiPAP initiated (IPAP 12/EPAP 6), ABG PaO2/FiO2 220 mmHg
6-12 Hours (Early Improvement)	**Vital signs:** HR 95 bpm, BP 105/70 mmHg (norepi wean), RR 22, SpO2 94% on BiPAP **Respiratory status:** Reduced crackles, less dyspnea **Hemodynamic status:** Norepinephrine tapered to 2 mcg/min **Clinical notes:** CXR shows persistent but less dense infiltrates, BNP 85 pg/mL normal
24 Hours (Significant Recovery)	**Vital signs:** HR 88 bpm, BP 118/75 mmHg (off norepi), RR 18, SpO2 96% on 2L NC **Respiratory status:** Minimal crackles, comfortable breathing **Hemodynamic status:** Fully hemodynamically stable **Clinical notes:** BiPAP to high-flow nasal cannula, PaO2/FiO2 >300 mmHg
48 Hours (Near Recovery)	**Vital signs:** HR 82 bpm, BP 125/78 mmHg, RR 16, SpO2 98% RA **Respiratory status:** Clear lung sounds **Hemodynamic status:** Normal **Clinical notes:** CXR nearly normal, transferred from ICU to ward
Discharge (Day 5)	**Vital signs:** HR 78 bpm, BP 128/80 mmHg, RR 16, SpO2 99% RA **Respiratory status:** Normal respiratory exam **Hemodynamic status:** Stable **Clinical notes:** No residual symptoms, Hb 9.8 g/dL, discharged with GI follow-up

(**Abbreviations:** ABG: Arterial Blood Gas, BiPAP: Bilevel Positive Airway Pressure, BNP: B-type Natriuretic Peptide, BP: Blood Pressure, bpm: Beats per minute, CXR: Chest X-ray, EPAP: Expiratory Positive Airway Pressure, GI: Gastrointestinal, Hb: Hemoglobin, HR: Heart Rate, IPAP: Inspiratory Positive Airway Pressure, mcg/min: Micrograms per minute, mmHg: Millimeters of mercury, NC: Nasal Cannula, Norepi: Norepinephrine, NRB: Non-rebreather Mask, PaO2/FiO2: partial pressure of oxygen, pg/mL: Picograms per milliliter, RA: Room Air, RR: Respiratory Rate, SpO_2: Peripheral Capillary Oxygen Saturation)

FOLLOW UP

Following the acute resolution of TRALI and the patient's discharge on Day 5, a comprehensive long-term management plan is paramount to address the root cause of anemia and prevent a recurrence of transfusion-related complications. The goal is to correct the underlying iron deficiency anemia (IDA) without relying on further blood products, which now pose a higher risk to this patient. The primary strategy involves aggressive treatment of iron deficiency anemia (Hb 9.8 g/dL; Ferritin 8 ng/mL) through high-dose oral Ferrous Sulfate (325 mg) supplemented with Vitamin C and Avoid calcium or antacids within 2 hours of the dose ,or a transition to IV iron infusions (Ferric Carboxymaltose) if oral adherence remains poor. Furthermore, the patient's medical records have been flagged with a permanent TRALI alert to ensure that any future unavoidable transfusions utilize leukoreduced or washed products. Simultaneously, the hospital laboratory has initiated a "lookback" investigation to screen the implicated donor for HLA and HNA antibodies, ensuring that the specific donor is deferred from future plasma-rich donations to safeguard the broader patient population.

DISCUSSION

In 1985, the formal diagnostic criteria for TRALI were established, which have been extensively utilised in subsequent years to differentiate this lethal reaction from other forms of acute respiratory distress. The diagnosis of TRALI relies on a strict temporal relationship, with onset within six hours of transfusion and the clinical evidence of non-cardiogenic pulmonary oedema. The present case report is supported by the widely accepted "Two-Hit" mechanism, where the patient's underlying inflammatory state served as the "First Hit," priming neutrophils that were subsequently activated by the "Second Hit" of transfused blood components.

Current management evidence emphasises that prompt recognition and aggressive supportive care are the cornerstones of reducing the 5 to 25% mortality rate associated with this condition. This case aligns with standard protocols that prioritize respiratory and hemodynamic support while strictly avoiding interventions like diuretics, which are indicated for TACO but are harmful in the setting of TRALI, stated in the case study by Singh et al. (2023). As observed in this patient's 48-hour recovery timeline, the clinical course of TRALI is often acute but reversible with appropriate high-acuity nursing and medical management, leading to full recovery without residual deficits. These findings reinforce the importance of national blood bank mitigation strategies, such as male-only plasma donor selection and universal leukoreduction, which have significantly lowered the global incidence of this leading cause of transfusion-related fatality.[5-7]

PREVENTION

The primary strategy for preventing TRALI focuses on mitigating the "Second Hit" from the transfused blood product, mainly by managing donor selection. To prevent Immune TRALI, blood banks prioritize using male-only plasma for high-plasma-volume components (like Fresh Frozen Plasma) and screen female donors who have been pregnant for Anti- HLA and Anti -HNA antibodies, deferring those who test positive. To prevent Non-Immune TRALI, the universal practice of pre-storage leukoreduction is used to filter out donor white blood cells, thereby minimizing the accumulation of harmful biologically active lipids that activate neutrophils during blood storage. Together, these measures have significantly reduced the incidence of TRALI.[8,9]

DIFFERENTIATING TRALI FROM TACO: A CRITICAL DIAGNOSTIC CHALLENGE:

The most critical challenge in the emergency setting is differentiating between TRALI and TACO. It is important to differentiate

between TRALI and TACO because their underlying mechanisms and treatments are fundamentally opposite, as detailed in Table 2. Misdiagnosis and inappropriate treatment can lead to clinical deterioration and increased mortality. Accurate differentiation ensures timely and correct management, optimising patient outcomes in the emergency setting.[10,11]

Table 2: Misdiagnosis and inappropriate treatment can lead to clinical deterioration and increased mortality. Accurate differentiation ensures timely and correct management, optimising patient outcomes in the emergency setting[10,11]

Feature	Trali (Non-Cardiogenic)	Taco (Cardiogenic)
Pathophysiology	Immune-mediated capillary leak, increased permeability	Volume overload, increased hydrostatic pressure
Onset	Acute, usually within 6 hours of transfusion	Gradual, up to 12 hours post-transfusion
Blood Pressure	Hypotension	Hypertension or normal
Body Temperature	Fever is frequently present	Typically afebrile
BNP Levels	Normal or mildly elevated	Markedly elevated
Cardiogenic Signs	JVD & S3 Gallop Absent	JVD & S3 Gallop Present
Chest X-ray	New, bilateral, diffuse pulmonary infiltrates with a normal cardiac silhouette	Pulmonary edema with an enlarged heart (cardiomegaly) and pleural effusions
Response to Diuretics	No improvement, may worsen	Rapid improvement
Primary Treatment	Respiratory support, vasopressors, avoid diuretics	Diuretics, fluid restriction
Fluid Balance	No fluid overload	Positive fluid balance

Abbreviations: TACO: Transfusion-Associated Circulatory Overload, BNP: B-type Natriuretic Peptide, JVD: Jugular Venous Distention, S3: Third Heart Sound (S3 Gallop), CXR: Chest X-ray

ROLE OF HEALTH CARE PROFESSIONALS

The management of TRALI is an urgent, multidisciplinary effort where the survival of the patient hinges on coordinated action between nursing, laboratory, and health care professionals . Nurses act as the first line of defense, providing essential bedside vigilance to detect abrupt changes such as sudden dyspnea or fever and immediately halting the transfusion to prevent further pulmonary damage. Laboratory technicians and blood bank professionals serve as precision navigators, delivering the paramount rapid turnaround of ABGs and BNP levels that allow the team to differentiate TRALI from TACO, while later conducting a screenings for HLA/ HNA antibodies to safeguard future recipients and also by implementing donor screening strategies such as prioritizing male or nulliparous female donors. Finally, healthcare professionals lead the strategic response by prioritizing hemodynamic support over harmful diuretics and transitioning the patient to safer alternatives like IV iron therapy and GI investigations. Together, this synergy of early identification, diagnostic accuracy, and targeted management ensures a rapid recovery and prevents the recurrence of this life-threatening reaction.[12,13]

CONCLUSION

In conclusion, this case underscores that Transfusion-Related Acute Lung Injury (TRALI) remains a high-stakes clinical emergency requiring a vigilant, multidisciplinary approach for successful resolution. Healthcare providers ability to distinguish TRALI from TACO through physical exams and biomarkers is essential to avoid the potentially fatal administration of diuretics in a hemodynamically unstable patient. Ultimately, the synergy between nursing vigilance, laboratory precision, and informed clinical management is ensures patient safety and shifts the prognosis from life-threatening to a full recovery.

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