e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(10); 817-821

Original Research Article

Suspected Transfusion-Related Acute Lung Injury after Cesarean Delivery for Placenta Previa with Antepartum Hemorrhage: A Case Report

Sunita[1], Aashita Goyal[2], Richa Saroa[3], Sanjeev Palta[4]

[1]Department of Anaesthesia and Critical Care Institute- Pt. BD Sharma, PGIMS, Rohtak, Haryana, India
[2,3,4]Department of Anaesthesiology, Government Medical College and Hospital, Sector-32, Chandigarh, India

Received: 01-07-2026 / Revised: 15-08-2026 / Accepted: 21-09-2026
Corresponding author: Dr. Sunita
Conflict of interest: Nil

Abstract

Background: Transfusion-related acute lung injury (TRALI) is a leading cause of transfusion-related death, yet it is under-recognized in obstetric hemorrhage, where it overlaps clinically with transfusion-associated circulatory overload (TACO).

Case Presentation: An unbooked 33-year-old woman (G3P2L2) at 32 weeks' gestation presented with painless antepartum hemorrhage due to placenta previa and hemoglobin of 6.2 g/dL. She underwent emergency cesarean delivery under general anesthesia and received packed red cells, fresh frozen plasma and cryoprecipitate. Within 3 hours of transfusion, she developed fever, hypotension, tachycardia and hypoxemia (PaO2/FiO2 200), with bilateral upper- and mid-zone pulmonary infiltrates.

Management and Outcome: She received oxygen, vasopressors, corticosteroids, cautious diuresis and antibiotics; infective work-up was negative. She was off oxygen by day 5 and discharged on day 7 with a healthy neonate.

Conclusion: Hypoxemia within 6 hours of transfusion in obstetric hemorrhage warrants structured evaluation for TRALI versus TACO and prompt reporting to the transfusion service.

Keywords: Antepartum Hemorrhage, Blood Transfusion, Placenta Previa, Transfusion-Associated Circulatory Overload, Transfusion-Related Acute Lung Injury.

DOI: 10.25258/ijcpr.18.10.113

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Transfusion-related acute lung injury (TRALI) is acute hypoxemic respiratory failure due to non-cardiogenic (permeability) pulmonary edema that develops during or within 6 hours of transfusion of a blood component.[1] Together with transfusion-associated circulatory overload (TACO), it is a leading cause of transfusion-related death, and the two syndromes are difficult to separate at the bedside because both present with hypoxemia and bilateral pulmonary infiltrates.[2]

In a prospective study at two academic centers, the incidence of TRALI fell to approximately 0.8 per 10,000 units transfused (about 1 in 12,000 units) after transfusion of plasma from female donors was reduced,[3] and reported mortality ranges from 5% to 25%.[4] Women transfused for obstetric hemorrhage appear to be at particular risk: TRALI was identified in nearly one-fifth of patients who received three or more red cell units for postpartum hemorrhage in one series,[5] and sequential TACO and TRALI have been described in the postpartum period.[6] Placenta previa, the implantation of the placenta over or near the internal os in the lower uterine segment, has a pooled worldwide prevalence of 5.2 per 1,000 pregnancies, with the highest prevalence in Asian studies (12.2 per 1,000).[7] It typically presents with painless antepartum hemorrhage that may necessitate urgent delivery and transfusion. We report a woman with placenta previa and severe antepartum hemorrhage who developed acute hypoxemia, hypotension and bilateral pulmonary infiltrates after red cell transfusion, and discuss how TRALI can be distinguished from TACO and other causes in this setting.

Case Report

Presentation: A 33-year-old woman, gravida 3, para 2, living 2 (G3P2L2), at 32 weeks of gestation presented to the obstetric emergency department with sudden-onset, painless bleeding per vaginum of 2 hours' duration. The bleeding was fresh, with clots, began at rest and increased after a vaginal

examination, and was accompanied by reduced fetal movements. She had one episode of spotting a month earlier for which she had not sought care. The pregnancy was spontaneously conceived and unbooked, with no antenatal investigations; gestational age was calculated from the last menstrual period. Both previous children had been delivered vaginally at term at home without antenatal care. There was no history of uterine surgery, trauma, hypertension, diabetes mellitus, asthma, thyroid disease, tuberculosis, seizure disorder or previous blood transfusion.

On examination, she was conscious and oriented, thinly built (weight 45 kg) and afebrile, with a pulse rate of 70/min, blood pressure of 96/64 mm Hg, respiratory rate of 18/min and marked pallor. There was no jugular venous distension, and the chest and cardiovascular examinations were normal. The uterus corresponded to 34 weeks, was non-tender, and the fetus was cephalic. On speculum examination, approximately 100 mL of clots were removed and bleeding was seen through the cervical os. Ultrasonography showed a posterior placenta completely covering the internal os, a fetal heart rate of 120/min and a biophysical profile score of 8/8. Hemoglobin was 6.2 g/dL. A diagnosis of antepartum hemorrhage due to major placenta previa with severe anemia was made.

Surgery and Anesthesia: Emergency lower segment cesarean section was undertaken for fetal bradycardia (fetal heart rate 80/min). After aspiration prophylaxis, rapid sequence induction was performed with thiopentone 3 mg/kg and succinylcholine 1.5 mg/kg, and the trachea was intubated with a 6.5-mm cuffed endotracheal tube.

Anesthesia was maintained with sevoflurane 1%–1.5% in oxygen and air (50:50) with volume-controlled ventilation, and fentanyl 1.5 µg/kg was given after delivery. Intraoperative hypotension required a noradrenaline infusion (0.05 µg/kg/min) through an external jugular venous cannula, which was weaned before the end of surgery. Estimated blood loss was about 1 L and urine output 100 mL; 1,000 mL of crystalloid, 500 mL of colloid and one unit of packed red blood cells (PRBC) were infused. A live female infant weighing 2.38 kg was delivered, with Apgar scores of 7 and 9 at 1 and 5 minutes. The patient was extubated at the end of surgery and transferred to the high-dependency unit (HDU).

Postoperative Course: In the HDU, a second PRBC unit was transfused (post-transfusion hemoglobin 7.6 g/dL), followed by a third. On the day of surgery, she developed fever up to 39°C, which was treated with paracetamol; it was not investigated as a possible febrile non-hemolytic transfusion reaction. After the fever settled, a fourth PRBC unit was transfused, followed by 10 mL of 10% calcium gluconate given intravenously over 10 minutes. PRBC and fresh frozen plasma were transfused in a 1:1 ratio, together with cryoprecipitate, and the total recorded fluid intake on the day of surgery was 2,660 mL.

Two to three hours after the fourth unit, she developed fever (39.4°C), tachypnea (28/min), tachycardia (125/min), hypotension (70/41 mm Hg) and hypoxemia (SpO2 88% on room air), with bilateral coarse crepitations. Arterial blood gas analysis showed metabolic acidosis with hypoxemic respiratory failure: pH 7.28, PaCO2 25 mm Hg, lactate 4 mmol/L and a PaO2/FiO2 ratio of 200 on FiO2 1.0. Chest radiography showed bilateral infiltrates, predominantly in the upper and mid zones, with clear costophrenic angles [Figure 1]. Electrocardiography showed sinus tachycardia, and screening transthoracic echocardiography showed a left ventricular ejection fraction (LVEF) of 50%–55% with trivial mitral regurgitation.

Oxygen was given through a Venturi mask (FiO2 0.6) and escalated to a non-rebreathing mask at 15 L/min. A noradrenaline infusion was used to maintain a mean arterial pressure above 65 mm Hg, and vasopressin (2.4 U/h, 0.04 U/min) was added on postoperative day 1. She received nebulized salbutamol with ipratropium bromide every 6 hours and budesonide every 12 hours, single intravenous doses of hydrocortisone 100 mg and dexamethasone 6 mg, and intravenous furosemide 10 mg once the blood pressure permitted. Low-molecular-weight heparin 40 mg subcutaneously once daily was started for thromboprophylaxis. Empirical intravenous ceftriaxone, metronidazole and gentamicin, with oral doxycycline, were started; after the febrile episode, the intravenous antibiotics were changed to meropenem on postoperative day 1.

Opinions from pulmonary medicine and cardiology were obtained; the initial impression of the pulmonary medicine team was sepsis with septic shock and acute respiratory distress syndrome (ARDS), and they advised avoiding fluid overload and keeping a low threshold for intubation with low tidal volume ventilation. The reaction was reported to the blood bank, repeat blood grouping and cross-matching were performed, and the direct antiglobulin test was negative; donor antibody testing was not done.

Serial laboratory results are summarized in [Table 1]. On postoperative day 1, there was neutrophilic leukocytosis (31.4 × 10^9/L, 92% neutrophils), and C-reactive protein rose from 5.5 mg/L on admission to 244 mg/L on postoperative day 2, with serum albumin of 2.5 g/dL.

Tests for malaria, dengue, scrub typhus and typhoid (Widal test) were negative, as were blood culture,

urine culture and a high vaginal swab, including a potassium hydroxide preparation.

Ultrasonography showed no retained products of conception, no intra-abdominal collection and no free fluid in the hepatorenal pouch, with small bilateral pleural effusions (pleural separation 13 mm on the right and 17 mm on the left). Echocardiography on postoperative day 1 again showed an LVEF of 50%–55% with trivial mitral regurgitation, but on postoperative day 3 it showed global left ventricular hypokinesia with moderate systolic dysfunction (LVEF 30%–35%), mild mitral and trivial tricuspid regurgitation, and no pericardial effusion, intracardiac clot or vegetation. She was off oxygen by postoperative day 5, when a repeat chest radiograph showed no infiltrates, and she was discharged on day 7 breathing room air, with a healthy neonate. The sequence of events is summarized in [Table 2].

Table 1: Serial laboratory investigations

Parameter (unit)	Day 0	POD 1	POD 2
Hemoglobin (g/dL)	—	10.8	—
Hematocrit (%)	—	33	—
Total leukocyte count (×10⁹/L)	—	31.4	—
Neutrophils / lymphocytes (%)	—	92.1 / 3.4	—
Platelet count (×10⁹/L)	—	280	—
Sodium (mEq/L)	128	—	140
Potassium (mEq/L)	3.9	—	3.5
Chloride (mEq/L)	100	—	107
Urea (mg/dL)	17	—	32
Creatinine (mg/dL)	0.7	—	0.6
Uric acid (mg/dL)	4.3	—	5.4
Calcium (mg/dL)	9.2	—	8.1
Phosphorus (mg/dL)	3.4	—	2.9
Total bilirubin (mg/dL)	0.6	—	0.6
AST (IU/L)	55	—	46
ALT (IU/L)	47	—	34
ALP (IU/L)	200	—	133
Total protein (g/dL)	6.7	—	5.5
Albumin (g/dL)	—	—	2.5
C-reactive protein (mg/L)	5.5	—	244
LDH (IU/L)	218	—	419

Day 0: Day of admission and surgery, POD: Postoperative day, —: Not done, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, LDH: Lactate dehydrogenase

Table 2: Timeline of clinical events

Time	Events
Day 0 (admission)	Painless antepartum hemorrhage at 32 weeks (by last menstrual period); hemoglobin 6.2 g/dL; placenta completely covering the internal os
Day 0 (surgery)	Emergency cesarean section under general anesthesia for fetal bradycardia; noradrenaline 0.05 µg/kg/min; 1,000 mL crystalloid, 500 mL colloid and one PRBC unit; female infant, 2.38 kg, Apgar 7 and 9
Day 0 (HDU)	PRBC units 2 and 3 (PRBC and FFP in a 1:1 ratio, with cryoprecipitate); fever 39°C; PRBC unit 4
Day 0, 2–3 h after unit 4	Fever 39.4°C, RR 28/min, HR 125/min, BP 70/41 mm Hg, SpO2 88% on room air, PaO2/FiO2 200; bilateral upper- and mid-zone infiltrates; LVEF 50%–55%; oxygen, noradrenaline, corticosteroids, furosemide; reaction reported to blood bank
Postoperative day 1	Pulmonary medicine review; vasopressin added; antibiotics changed to meropenem; TLC 31.4 × 10⁹/L; ABG pH 7.347, lactate 2.3 mmol/L; LVEF 50%–55%
Postoperative day 2	C-reactive protein 244 mg/L
Postoperative day 3	Global left ventricular hypokinesia, LVEF 30%–35%
Postoperative day 5	Off oxygen; chest radiograph clear of infiltrates
Postoperative day 7	Discharged on room air with a healthy neonate

PRBC: Packed red blood cells, FFP: Fresh frozen plasma, HDU: High-dependency unit, RR: Respiratory rate, HR: Heart rate, BP: Blood pressure, LVEF: Left ventricular ejection fraction, ABG: Arterial blood gas

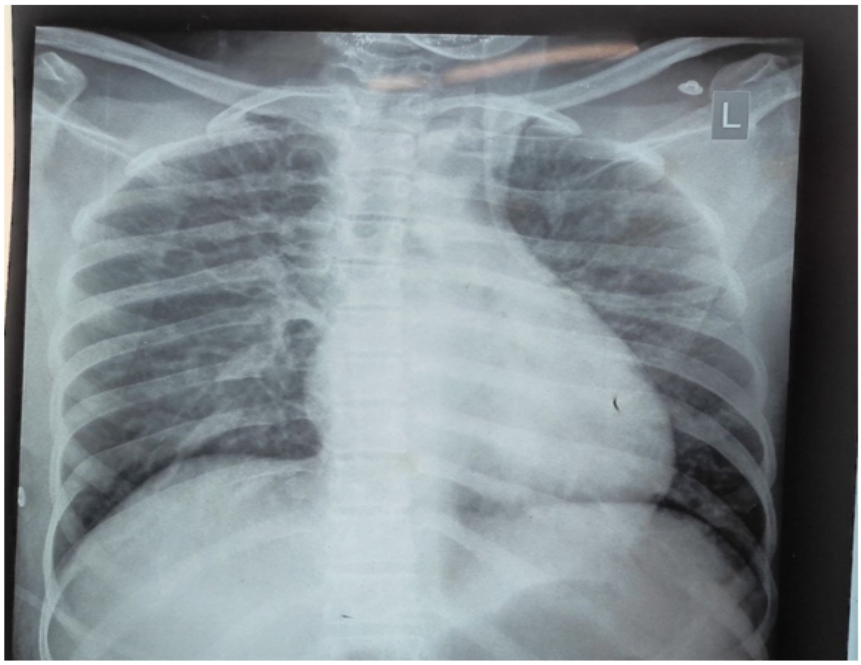


Figure 1: Anteroposterior chest radiograph obtained in the high-dependency unit on the day of surgery, showing bilateral infiltrates, predominantly in the upper and mid zones, with clear costophrenic angles

Discussion

The 2019 consensus redefinition classifies TRALI into two types.[1] TRALI type I applies to patients without a risk factor for ARDS who develop, during or within 6 hours of transfusion, acute hypoxemia (PaO2/FiO2 ≤300 mm Hg or SpO2 <90% on room air) with clear bilateral pulmonary edema on imaging and no evidence of left atrial hypertension, or left atrial hypertension that is judged not to be the main contributor.

TRALI type II applies when the same features occur in a patient with an ARDS risk factor, or mild pre-existing ARDS, whose respiratory status was stable in the 12 hours before transfusion. The term "possible TRALI" has been dropped; cases that cannot be separated from circulatory overload are reported as "TRALI/TACO cannot distinguish". TRALI remains a clinical diagnosis, and detection of cognate leukocyte antibodies is not required.[1]

Our patient developed acute hypoxemia (PaO2/FiO2 200) with bilateral infiltrates 2–3 hours after transfusion of red cells together with plasma-rich components (fresh frozen plasma and cryoprecipitate), which are most often implicated in TRALI.[2] Fever and hypotension rather than hypertension, a preserved LVEF on two echocardiograms around the time of the event, and clearing of the infiltrates within 5 days also favor TRALI. However, she also had ARDS risk factors, namely hemorrhagic shock requiring vasopressors and multiple transfusions, and fever with marked leukocytosis and a C-reactive protein of 244 mg/L led the pulmonary medicine team to suspect septic shock with ARDS, although the infective work-up was negative. Because her respiratory status was stable before transfusion (SpO2 96% on room air before surgery, extubated after surgery), the presentation best fits TRALI type II. The pathophysiology is generally explained by a two-hit model, in which the patient's clinical condition primes pulmonary neutrophils and antibodies or other mediators in the transfused product provide the second hit.[2]

TACO must be actively excluded. The revised international surveillance definition requires onset within 12 hours of transfusion and at least three criteria, including acute or worsening respiratory compromise and/or radiological pulmonary edema, together with cardiovascular changes, evidence of fluid overload or a raised brain natriuretic peptide (BNP).[8] Several features in our patient are consistent with circulatory overload: about 2.7 L of fluid, including PRBC and fresh frozen plasma in a 1:1 ratio, on the day of surgery in a 45-kg woman with hypoalbuminemia (2.5 g/dL), small bilateral pleural effusions on ultrasonography, tachycardia and a subsequent fall in LVEF to 30%–35%. Natriuretic peptides were not measured, so a contribution from circulatory overload cannot be fully excluded. Haldar and Samanta described a woman with postpartum hemorrhage who developed TACO followed by TRALI, showing that the two can coexist or follow each other in the same patient.[6]

The new left ventricular dysfunction also requires consideration of peripartum cardiomyopathy, which typically presents as heart failure with reduced ejection fraction in the last month of

pregnancy or in the months after delivery in women without another cause of heart failure; natriuretic peptides, electrocardiography and echocardio graphy are recommended for diagnosis. [9] Septic or stress-related myocardial dysfunction is another possibility. Echocardiography was not repeated before discharge, so recovery of left ventricular function could not be confirmed, and follow-up echocardiography is advisable.

Management of TRALI is supportive: stopping the transfusion, oxygen, ventilatory support when needed and hemodynamic support. No specific therapy has proven benefit, and diuretics, which are appropriate for TACO, may worsen hypotension in TRALI.[2,4] Corticosteroids, used in our patient, have no established role. Prevention depends on donor strategies; reducing transfusion of plasma from female donors was associated with a fall in TRALI incidence,[3] and every suspected case should be reported to the transfusion service so that implicated donors can be investigated. The strengths of this report are the documented timing of events, serial echocardiography and systematic exclusion of infection. Its limitations are the absence of donor antibody testing and natriuretic peptide measurement, and the lack of investigation of the earlier febrile episode as a transfusion reaction.

Conclusion

New hypoxemia during or within 6 hours of transfusion in a woman with obstetric hemorrhage should prompt immediate cessation of the transfusion and a structured evaluation for TRALI versus TACO, including the timing of onset, fluid balance, natriuretic peptides and echocardiography. Classifying such events using current consensus definitions and reporting them to the transfusion service allows donor investigation and helps prevent recurrence.

References

1. Vlaar APJ, Toy P, Fung M, Looney MR, Juffermans NP, Bux J, et al. A consensus redefinition of transfusion-related acute lung injury. Transfusion 2019;59:2465-76.
2. Semple JW, Rebetz J, Kapur R. Transfusion-associated circulatory overload and transfusion-related acute lung injury. Blood 2019;133:1840-53.
3. Toy P, Gajic O, Bacchetti P, Looney MR, Gropper MA, Hubmayr R, et al. Transfusion-related acute lung injury: Incidence and risk factors. Blood 2012;119:1757-67.
4. Yu Y, Lian Z. Update on transfusion-related acute lung injury: An overview of its pathogenesis and management. Front Immunol 2023;14:1175387.
5. Teofili L, Bianchi M, Zanfini BA, Catarci S, Sicuranza R, Spartano S, et al. Acute lung injury complicating blood transfusion in post-partum hemorrhage: Incidence and risk factors. Mediterr J Hematol Infect Dis 2014;6:e2014069.
6. Haldar R, Samanta S. Post-partum sequential occurrence of two diverse transfusion reactions (transfusion associated circulatory overload and transfusion related acute lung injury). J Emerg Trauma Shock 2013;6:283-6.
7. Cresswell JA, Ronsmans C, Calvert C, Filippi V. Prevalence of placenta praevia by world region: A systematic review and meta-analysis. Trop Med Int Health 2013;18:712-24.
8. Wiersum-Osselton JC, Whitaker B, Grey S, Land K, Perez G, Rajbhandary S, et al. Revised international surveillance case definition of transfusion-associated circulatory overload: A classification agreement validation study. Lancet Haematol 2019;6:e350-8.
9. 9. Bauersachs J, König T, van der Meer P, Petrie MC, Hilfiker-Kleiner D, Mbakwem A, et al. Pathophysiology, diagnosis and management of peripartum cardiomyopathy: A position statement from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. Eur J Heart Fail 2019;21:827-43.