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CASE REPORT

Transfusion-related acute lung injury and treatment with high-flow oxygen therapy in a pediatric patient: a case report

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KEYWORDS
Transfusion-related acute lung injury;
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Abstract Transfusion-Related Acute Lung Injury (TRALI) is an immune-inflammatory lung pathology that manifests within the first 6–72 hours after administration of blood products. However, due to reduced awareness of TRALI, it continues to be an underreported and often underdiagnosed complication of transfusion therapy. We report a case of a 6-year-old girl with myelodysplastic syndrome and TRALI developed in the first hour after platelet transfusion. Diagnosis of TRALI is based on the exclusion of etiologic factors such as volume overload and cardiogenic pulmonary edema following transfusion. Symptoms responded to high-flow oxygen therapy, so intubation was not attempted and full recovery was achieved.

Introduction

Transfusion-related acute lung injury (TRALI) is an immune-inflammatory lung pathology that manifests with tachycardia, tachypnea, dyspnea, hypoxia, and bilateral pulmonary edema within the first 6–72 hours after administration of blood products. Symptoms such as pinkish secretion, hypotension, fever, and cyanosis are seen in the respiratory tract.[1] It is the main cause of death from transfusion and the mortality rates range between 6–14 percent.[1,2]

Transfusion-related acute lung injury and transfusion-associated circulatory overload (TACO) are acute respiratory distress syndromes that occur within 6 hours after blood transfusion. It is very difficult to distinguish TRALI and TACO from the underlying causes of lung injury, fluid overload, or from each other. TACO is characterized by pulmonary hydrostatic (cardiogenic) edema, while TRALI manifests as non-cardiogenic permeability pulmonary edema.[2-4]

In this case report, a high-flow oxygen therapy (HFOT) treatment of a pediatric patient who developed TRALI after platelet transfusion was discussed.

Abbreviations: TRALI, Transfusion-Related Acute Lung Injury; TACO, Transfusion-Associated Circulatory Overload; HFOT, High-Flow Oxygen Therapy; PEEP, Positive end-Expiratory Pressure; FiO2, Fraction of inspired Oxygen; PACU, Postanesthesia Care Unit; PaO2, Partial pressure of Arterial Oxygen; PaCO2, Partial pressure of Arterial Carbon dioxide; SO2, Arterial Oxygen Saturation.

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Table 1 Postoperative blood gas analyses of the patient.

Postoperative	20th min	2nd h	6th h	10th h	24th h	48th h
PH	7.31	7.34	7.28	7.37	7.43	7.43
$PaCO_2$ (mmHg)	44	41.5	47	41.3	38.2	39.3
PaO_2 (mmHg)	56.8	64.3	89.8	80.9	72.1	124
SO_2 (%)	82.6	88	96.1	95.8	94.5	99
Lactate ($mmoL.L^{-1}$)	1.7	1.7	1.5	1.9	1.7	1.1
PaO_2/FiO_2	113	107	150	162	160	276
Blood pressure (mmHg)	85/40	96/50	95/52	92/45	90/48	94/46
Heart rate (bpm)	145	125	120	118	112	108
Respiratory rate (min^{-1})	45	28	26	24	20	20
Fluid balance (mL)	+520	+170	+10	-150	-200	-250
Temperature (°C)	36.2	36.3	36.6	36.8	36.7	36.6
Therapy	Mask	HFOT	HFOT	HFOT	HFOT	HFOT

Mask, Simple Face Mask (5 $L.min^{-1}$ O_2); HFOT, High-Flow Oxygen Therapy; min, minute; h, hour; PaO_2, Partial pressure of Arterial Oxygen; $PaCO_2$, Partial pressure of Arterial Carbon dioxide; SO_2, Arterial Oxygen Saturation.

Case report

A 6-year-old girl (17 kg, 110 cm) was admitted for splenorenal shunting due to the development of portal hypertension. Her medical history revealed myelodysplastic syndrome and upper gastrointestinal bleeding five times due to esophageal varices. Her hemogram values were as follows: leukocyte: 1.920 mm^{-3}, hematocrit: 25.9%, and platelet: 45.000 mm^{-3}. Since a shunt operation was planned for the patient without active bleeding, preoperatively platelet transfusion was not indicated. The patient was given midazolam (0.5 $mg.kg^{-1}$ oral) for premedication and general anesthesia was performed under standard anesthesia monitoring including electrocardiogram, pulse oximetry, noninvasive blood pressure, and axillary temperature (basal arterial blood pressure: 92/42 mmHg, heart rate: 108 bpm, respiratory rate: 22 min^{-1}, Sat: 96% in-room air, and temperature: 36.6°C). Atropine (20 $\mu g.kg^{-1}$), ketamine (0.5 $mg.kg^{-1}$), and rocuronium (0.6 $mg.kg^{-1}$) were administered to the patient after induction of anesthesia with 8% sevoflurane. Maintenance of anesthesia was provided with sevoflurane (0.5–1 MAC), remifentanil infusion (0.25–0.5 $\mu g.kg^{-1}.min^{-1}$), and rocuronium (0.15 $mg.kg^{-1}$) when necessary. Mechanical ventilation settings after intubation with volume control mode were as follows: 7 $mL.kg^{-1}$ tidal volume (120 mL), 24 min^{-1} respiratory rate, 5 cmH_20 positive end-expiratory pressure (PEEP), 50% Fraction of inspired Oxygen (FiO_2), peak pressure: 15 cmH_20, and plateau pressure: 12 cmH_20 (The Dräger Perseus A500, Dräger Medical, Lubeck, Germany). The fluid treatment was provided with 4/2/1 rule (Holliday and Segar formula) and 0.9% NaCl for the patient who had a preoperative fasting period of 8 hours. Platelet transfusion was performed at 10 $mL.kg^{-1}$ (at an infusion rate of 170 $mL.h^{-1}$), approximately in the middle of the operation due to low platelet count and bleeding in the form of leakage in the tissue. The operation time was 180 minutes and the patient was given 550 mL isotonic and 170 mL platelet solution. The amount of bleeding during the operation was 50 mL and the urine output was 150 mL. The patient, who underwent decurarization with sugammadex (4 $mg.kg^{-1}$), was taken to the postanesthesia care unit (PACU) after being extubated in the operating room (arterial blood pressure: 88/44 mmHg, heart rate: 118 bpm, respiratory rate: 25 min^{-1}, Sat: 95% in-room air, and temperature: 36°C). In PACU at 20 minutes, desaturation (Sat: 80%, in-room air) and respiratory distress developed in the patient (Table 1). The patient did not have a pink frothy airway secretion. Oxygen therapy (5 $L.min^{-1}$) was started with a simple face mask (non-breathing mask,

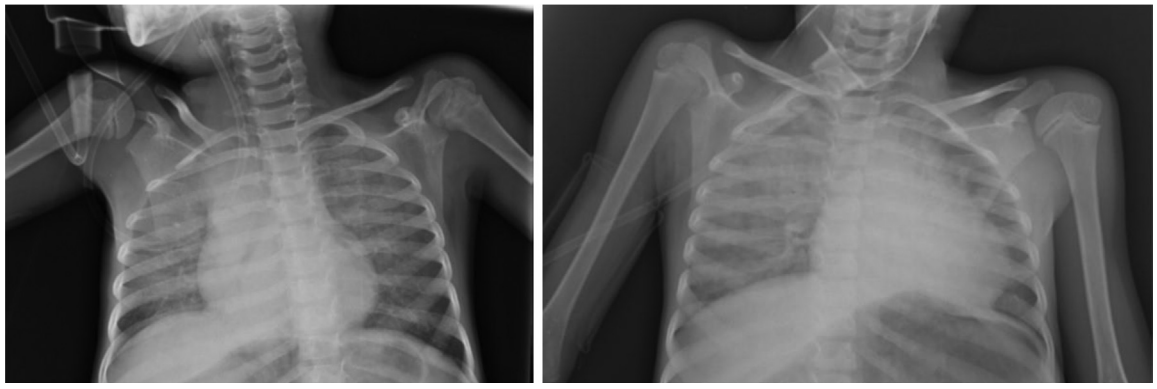

Figure 1 (a) Posterior-anterior chest radiography at the postoperative 20th minute. (b) Posterior-anterior chest radiography at the postoperative 2nd hour (after furosemide treatment was stopped).

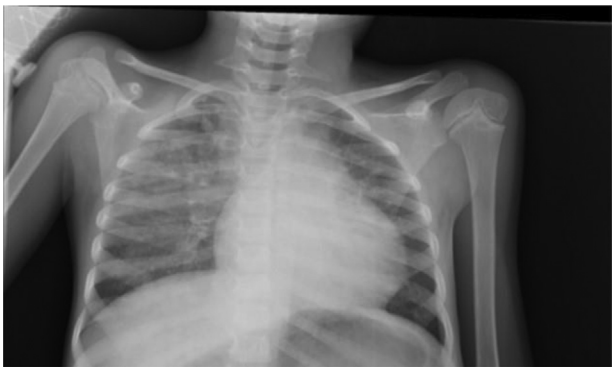

Figure 2 Posterior-anterior chest radiography at the postoperative 24th hour (high-flow oxygen therapy was being implemented).

Plastimed, Turkey). On the other hand, widespread crepitant rales were detected during auscultation of the lung. Pulmonary edema was confirmed by a chest X-Ray (Fig. 1a). Furosemide bolus was administered at a dose of 0.5 $mg.kg^{-1}$ with a preliminary diagnosis of pulmonary edema and the dose of the oxygen was increased to 10 $L.min^{-1}$. Within one hour after bolus injection of furosemide, 350 mL of urine diuresis was provided. Ejection fraction of the patient was 65% in transthoracic echocardiography performed by a pediatric cardiologist at the first hour in PACU, the patient had not demonstrated any evidence of cardiac failure and the diagnosis of TRALI was confirmed (Furosemide treatment was stopped) (Fig. 1b). There was no response to oxygen therapy delivered with the simple face mask (arterial blood pressure: 81/38 mmHg, heart rate: 142 bpm, respiratory rate: 44 min^{-1}, Sat: 85%); for this reason, HFOT (10 $L.min^{-1}$ flow, 60% FiO_2) was administered with a nasal cannula (Optiflow Junior 2 Nasal Interface, Fisher And Paykel Healthcare Limited (F&PH) Airvo2 HFNC High-flow oxygen therapy, Auckland, New Zealand) and the patient was transferred to the ICU. The postoperative fluid therapy was set to 1500 $mL.m^{-2}$ (Isolyte P in 5% Dextrose, Eczacibasi-Baxter, Istanbul, Turkey). Paracetamol (60 $mg.kg^{-1}.day^{-1}$) and morphine infusion (20 $\mu g.kg^{-1}.h^{-1}$) was started for analgesia. In the second hour after surgery, the ABG parametres of the patient were as follows: pH: 7.34, $PaCO_2$: 41.5 mmHg, PaO_2: 64.3 mmHg, SO_2: 88%. After these ABG parametres were obtained, the flow in HFOT was increased to 15 $L.min^{-1}$ and then parametres of blood gas analysis were determined as pH: 7.28, $PaCO_2$: 47 mmHg, PaO_2: 89.8 mmHg (Table 1). High-flow oxygen therapy was continued for three days and the flow rate and FiO_2 were adjusted for ABG results (Fig. 2 and 3, Table 1). The patient did not need an inotrope or a vasoconstrictor during intraoperative and postoperative periods. At the end of the third day, the patient was followed with a simple face mask ventilation (3–5 $L.min^{-1}$ O_2) for two days. The patient who was found to have the functional distal splenorenal shunt by ultrasound examination was discharged after being followed up in the pediatric surgery service for 5 days.

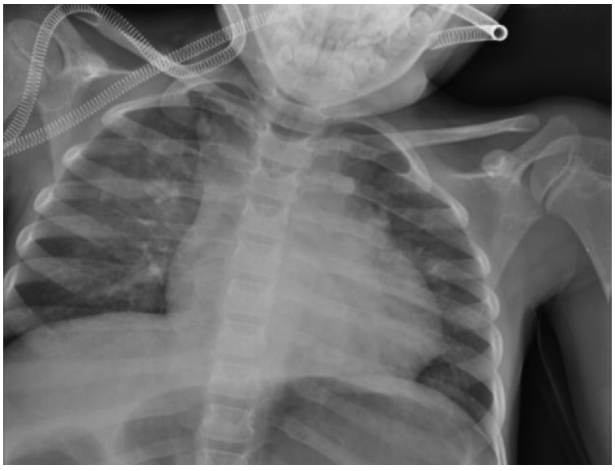

Figure 3 Postoperative 3rd day (after high-flow oxygen therapy).

Discussion

Transfusion-related acute lung injury is a complication of a serious transfusion of blood products and is the most common cause of transfusion-related deaths. Although its etiology and incidence are not fully known, it is estimated to be one in 5,000 blood product transfusions.[2] Patients with diagnosis of malignancy, shock, higher peak airway pressure during mechanical ventilation, positive intravascular fluid balance, low interleukin-10 levels, systemic inflammation, and those undergoing cardiovascular and liver surgery pose a risk for TRALI.[4] Our patient had myelodysplastic syndrome, so she had received transfusions many times within the last 2 years. Due to these factors, our patient was in the risk group for TRALI. However, in the preoperative period, she had not edema or rales and fluid therapy was not administered. High-pressure values were not applied in mechanical ventilation. Nevertheless, the patient's postoperative PaO_2/FiO_2 ratios showed that a moderate TRALI developed. In the intraoperative period, positive-pressure mechanical ventilation applied, and lack of increase in airway pressures may have masked TRALI symptoms.

Transfusion-associated circulatory overload, which develops after excessive blood volume, gives symptoms of dyspnea, tachypnea, hypoxemia, and jugular venous fullness and is the most commonly confused condition with TRALI. Diagnosis of TACO is made with left heart failure, high blood pressure, or tachycardia, and may include evidence of positive fluid balance or cardiogenic involvement. In contrast, TRALI is absolutely non-cardiogenic, there is no arterial hypertension and therefore volume overload should be excluded.[4] There is no specific treatment method for TRALI and transfusion should be terminated as soon as it is diagnosed. Fluid therapy and vasopressor agent support may be required in patients with hypotension. Therefore, it is recommended to avoid diuretics.[2,3]

In our patient, TRALI developed in the first hour after platelet transfusion, and manifested with dyspnea, tachypnea and desaturation. Hemodynamic instability and fever

were not detected. TRALI was confirmed by excluding excessive volume overload or TACO by transthoracic echocardiography. Therefore, diuretic treatment was stopped and fluid therapy was delivered at 1500 mL m^{-2}. Since there was no hypotension, there was no need for a vasoconstrictor. Despite diuretic treatment, the symptoms of the patient did not improve.

In TRALI, all patients need additional oxygen and more than 70% of patients need mechanical ventilation.[1] It has been found that HFOT can reduce the mortality of patients admitted to the intensive care unit due to acute respiratory failure compared to noninvasive ventilation. The beneficial effects of HFOT can be explained by its good tolerance and physiological properties such as high FiO_2, positive PEEP effect, and continuous dead space washout contributing to decreased work of breathing.[5]

Oxygen therapy and fluid restriction were applied primarily to our patient. Since oxygen therapy with the mask was inadequate we switched to HFOT. The patient's compliance to therapy was increased by using HFOT and both intubation and mechanical ventilation were avoided. If our patient had severe TRALI, intubation would have been inevitable. Physiological abnormalities and pulmonary infiltrations of TRALI usually improve in about 80% of patients in 4 days.[1] In our patient, the clinical symptoms and pulmonary infiltration resolved at the end of the 3rd day.

Conclusion

It should be kept in mind that TRALI may develop in patients with a history of transfusion. Because of vital importance in the treatment of hypoxemia while managing the patient, high-flow oxygen therapy, which is a well-tolerated noninvasive method, should be considered as an option in moderate or mild TRALI.

Informed consent

Informed consent from patient's family was received.

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Conflicts of interest

The authors declare no conflicts of interest.

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