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Respiratory Disease

Transfusion-Related Acute Lung Injury (TRALI) Type 1 in Ureteroscopy Procedure: A Case Report and Focused Review

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Abstract **Case Report**

Transfusion related lung injury is a life-threatening complication of transfusion. It is a rare condition but one of the leading causes of transfusion-related morbidity and mortality. We present a case of this infrequent complication in order to make it more familiar to clinicians.

Keywords: TRALI, Transfusion, Infiltrate, ARDS.

INTRODUCTION

Transfusion related lung injury is a life-threatening complication of transfusion. It is a rare condition but one of the leading causes of transfusion-related morbidity and mortality [1].

The incidence of this syndrome is estimated to 0,02% per unit transfused. However, the real prevalence remains undetermined because of the large number of misdiagnosed and unreported cases.

Diagnostic definition for this syndrome consists in hypoxia and bilateral pulmonary edema occurring during or within 6 hours of a transfusion, in the absence of cardiac failure or intravascular volume overload.

PATIENT AND OBSERVATION

A 27-year-old female patient (Height: 160 cm, weight: 63 kg) with a 5 years old history of renal lithiasis with failed treatment by extracorporeal lithotripsy procedure, admitted for ureteroscopy.

The patient was previously diagnosed and medicated for iron deficiency anemia. Blood and biochemistry tests done prior to the surgery were within normal limits. The chest radiography examination showed no special findings.

As the ureteroscopy was complicated with hemorrhage, the patient was transfused at the end of the procedure. Within 2 hours of blood transfusion, the patient's condition rapidly deteriorated with acute respiratory distress symptoms such as dyspnea, tachypnea and tachycardia. Oxygen saturation level decreased down to 87%. Blood pressure remained within normal range. On physical examination, the patient presented attenuation of breath sounds and diffuse crackles in the lungs. Chest X-ray and CT revealed bilateral diffuse pulmonary infiltrates (Fig 1). CT angiogram revealed the absence of pulmonary embolism. No relevant issues appeared in electrocardiogram and echocardiogram.

Laboratory investigations revealed leukocytosis (18960/L), Lymphopenia (830/L) and CRP level elevated up to 122mg/L.D-Dimers (<10000).

The clinical presentation, the absence of other pathological entities and the temporal relation with the blood transfusion suggested that the case met all TRALI syndrome criteria.

Oxygen therapy, methylprednisolone and antibiotics were administered, and the state of the patient improved within 24 hours without the need of respiratory ventilation support (Fig 2).

Table 1: The new consensus redefinition of diagnostic criteria

New consensus TRALI definition
TRALI Type 1: Patients who have no risk factors for ARDS and meet the following criteria: a) □ Acute onset □ Hypoxemia(P/F<300- or SpO2<90% on room air)* □ Clear evidence of bilateral pulmonary edema on imaging(e.g, chest radiograph, chest CT, or ultrasound) □ No evidence of LAH **,or if LAH is present ,it is judged to not be the main contributor to the hypoxemia b)Onset during or within 6 hours of transfusion *** c)no temporal relationship to an alternative risk factor for ARDS TRALI Type 2:Patients who have risk factors for ARDS(but who have not been diagnosed with ARDS) or who have existing mild ARDS(P/F of 200-300),but whose respiratory status deteriorates ****and is judged to be due to transfusion based on: a) Findings as described in categories a and b of TRALI type 1,and b) stable respiratory status in the 12 hours before transfusion
*If altitude is higher than 1000 m, the correction factor should be calculated as follows: [(P/F)x5barometric pressure/760)]. **Use objective evaluation when LAH is suspected (imaging e.g,echocardiography,or invasive measurement using e.g pulmonary artery catheter) ***Onset of pulmonary symptoms (e.g;,hypoxemia-lower P/F ratio or SpO2)should be within 6 hours of end of transfusion. The additional findings needed to diagnose TRALI(pulmonary edema on a lung imaging study and determination of lack of substantial LAH)would ideally be available at the same time but could be documented up to 24 hours after TRALI onset. ****Use P/F ratio deterioration along with other respiratory parameters and clinical judgment to determinate progression from mild to moderate or severe ARDS.

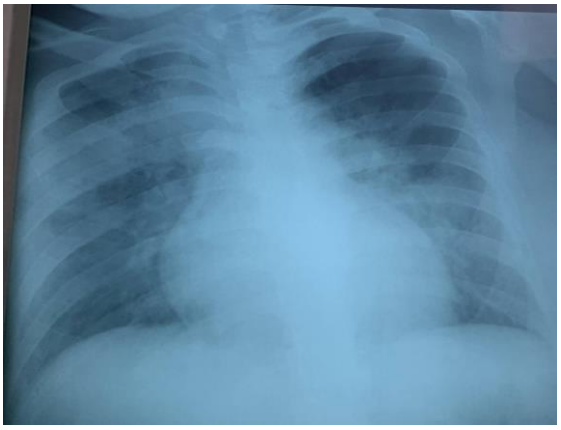

Figure 1: Supine Chest X-ray shows newly appeared bilateral pulmonary infiltrates

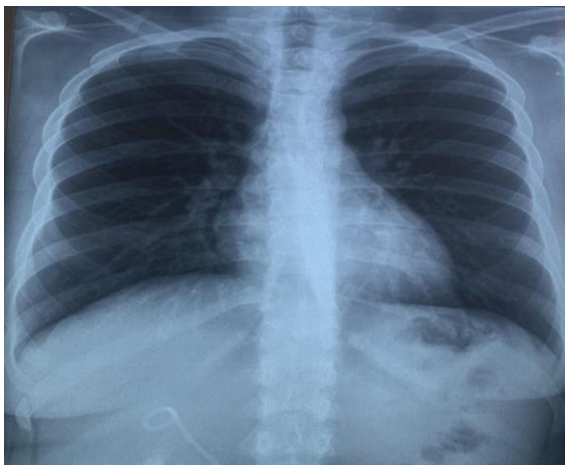

Figure 2: Chest X-ray 6 weeks after admission

DISCUSSION

Transfusion–related acute lung injury is a rare complication associated with transfusion of plasma products and packed red blood cells [2]. Reported more frequently in critically ill patients since they are transfused more commonly, the incidence of TRALI has been estimated between 0.02% and 0.05% per blood product transfused and between 0.04% and 0.16% per patient transfused [3, 4]. It is still nevertheless underestimated because of underdiagnosed and unreported cases [3].

The causes of TRALI are currently not fully understood but 2 main physiopathological forms are suggested:

1. "immune-mediated" TRALI, induced by activation of the recipient's neutrophils by alloantibodies in the donor blood product, leading to an inflammatory process causing lung injury.
2. "non-immune-mediated" TRALI, Inflammatory process caused by activation of biological compounds such as bioactive lipids or proinflammatory cytokines [5, 6].

The diagnosis of TRALI is primarily clinical and radiographic. The clinical presentation is similar to acute respiratory distress syndrome (ARDS). The symptoms occur commonly within 2 to 6 hours after the start of transfusion [7]. The most common symptoms are dyspnea, cough, fever and chills, tachypnea, tachycardia, cyanosis and cough. Both hypertension and hypotension have been reported [7, 8]. Physical

examination reveals diffuse crackles and breath sounds attenuation. The new consensus redefinition of diagnostic criteria are presented in Table I [9].

Radiographs of the chest reveal findings typical of ARDS. The alveolar and interstitial infiltrates are bilateral and diffuse, without cardiac enlargement or engorged pulmonary vessels. Echocardiography, pulmonary artery wedge pressure measurement and B serum natriuretic peptide (BNP) levels may be helpful in excluding hydrostatic and cardiogenic pulmonary edema. Arterial blood gas analysis shows hypoxia [3]. Laboratory findings may show hemoconcentration, leukopenia, neutropenia, hypocomplementemia, and hypoalbuminemia.

Suspected cases should be reported to blood transfusion centers for leukocyte antibody screening in recipient's and donor's blood [2]. Treatment is mainly symptomatic based on oxygen therapy. Mechanical ventilation is required in approximately 70% of cases [9]. The mortality rate for TRALI is about 16.3% [10]. Improvement is seen in most cases over 48-96 hours.

CONCLUSION

TRALI is a temporally related blood transfusion complication and a main cause of transfusion-related mortality, which may occur in the absence of an underlying condition. The diagnosis has become primarily clinical and radiographic, according to the new diagnostic criteria. They exclude nevertheless patients with preexisting lung injury. Treatment is mainly symptomatic and supportive; it includes oxygen supplementation and mechanical ventilation support.

Competing Interests: The authors declare no competing interest.

Authors' contributions

Souhail Raftani: Treating physician
Safaa Aidou: Treating physician
Hajar Arfaoui: Professor in charge
Hasna Jabri: Professor in charge
Wiam Elkhattabi: Professor in charge
Moulay Hicham Afif: Chief Professor

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