
Transfusion-Related Acute Lung Injury (TRALI) In A Multipara Patient

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Abstract

Introduction: Transfusion-Related Acute Lung Injury (TRALI) is a serious transfusion reaction in which HLA or leucocyte antigens present in the donor's serum thought to be responsible in the pathophysiology.

Case Report: Total knee prosthesis was placed into a 68 year old multipar patient who had controlled diabetes mellitus and hypertension and was assigned to the ASA II group. She had undergone left knee arthroscopy one year ago and had no transfusion history. No problems occurred after two units of blood transfusion in the perioperative period. During transfusion in the postoperative period respiratory distress, tachypnea and uneasiness occurred. Oxygen support with mask, 75 mg IV prednisolone, and antihistaminics were administered and the transfusion was discontinued. Arterial blood gases were as follows: pH: 7.31, paO_2 : 62mmHg, paCO_2 : 47.2mmHg, HCO_3 : 22.3mmol/L, BE: -3.9, O_2SAT : 86.1%. Chest x-ray showed infiltration. As the crossmatch was negative and the donor was her daughter we thought about a TRALI syndrome. Blood samples were taken for HLA and granulocyte specific antibody screening.

Discussion: In TRALI syndrome, the HLA or granulocyte specific antibodies (GSA) present in the donor blood react with the leukocyte antigens and activates the complement system of the recipient. Complement C5a causes neutrophil aggregation and sequestration in microvascular bed of lungs. Protease, acidic lipids and oxygen radicals released from neutrophils constitutes damage in pulmonary vascular area. Protein containing fluid leaks into alveolus and interstitium.

TRALI can be seen in a few minutes to 40 hours after blood or blood products transfusion. Clinically fever, shivering, tachycardia, cough and differing degrees of respiratory distress may be seen. Hypotension and urticaria may be accompanied. Generally, there is hypoxia in arterial blood gases (30-60 mmHg). Bilaterally pulmonary edema may be detected in the chest x-ray.

Respiratory support is essential in the management of TRALI. In 30% of the cases mask oxygen is sufficient and in 70% mechanical ventilation is necessary. Mortality rate is 5% in complications like pulmonary edema and pneumonia. With appropriate treatment patients can be improved without any sequels in following 48-96 hours. A great number of TRALI patients are multipara women. Antibody development usually occurs after recurrent blood transfusion and pregnancy. A person who has HLA or GSA should not be used as a donor in order to avoid a TRALI syndrome.

INTRODUCTION

TRALI is a severe transfusion reaction resulting in acute lung collapse damage due to blood transfusion. It is considered to be initiated by leucoagglutinins or HLA specific antibodies transfer which already existed in the donor serum. We wish to emphasize this syndrome in the example of our case in which acute formation of TRALI occurred on the second day postoperatively in a patient whose donor was her own daughter.

CASE REPORT

A sixty eight year old patient with a history of regular diabetes mellitus and hypertension presented for treatment. Because of her medical condition she was assigned to the ASA II group. She had no transfusion history and had an arthroscopy 2 years ago as a preparation to a total knee prosthesis because of gonarthrosis. Intraoperatively, 2 units of blood were given to the patient. The transfusion was stopped because of the development of respiratory distress on the 2nd postop day. Arterial blood gas analysis revealed marked hypoxemia and the chest X-ray showed new diffuse

bilateral alveolar infiltrates. The arterial blood parameters in the patient were as follows: pH: 7.312, paO_2 : 62mmHg, paCO_2 : 47.2mmHg, HCO_3 : 22.3mmol/L, BE: -3.9, O_2SAT : 86.1%. The patient had tachypnea and distress symptoms. Intravenous methylprednisolone (200 mgs) was applied to the patient immediately. Re cross-match was performed between donor and her own blood samples. No reaction was detected. The patient was diagnosed with TRALI because of no recross-match reaction, her history of being a multipara and the history of own daughter as her donor. The patient improved rapidly within 36 hours both in laboratory and physical examinations. The samples were sent to a more advanced centre for lymphocyte cross-match.

DISCUSSION

In most acute lung damage cases due to blood transfusion, passive transfer of donor neutrophils or HLA specific antibodies and activation of complementary cascade by reacting with host leukocyte antigens is the culprit. Within the activation of complementary cascade, C5a causes neutrophil aggregation and sequestration in the microvascular bed of the lung. The damage occurs due to secretion of proteases, acidic lipids and oxygen radicals by neutrophils. The liquid which includes proteases infiltrates the alveoli and interstitial areas.

TRALI may develop within few minutes to 40 hours after blood transfusion. Shivering, fever, tachycardia, coughing or respiratory distress can be seen in the physical examination. Hypotension and urinary reaction may accompany these symptoms. Usually, severe arterial hypoxia is found (30-50 mmHg). Bilateral lung edema can also be found.

Most cases of TRALI occur in multipara women. The antibodies in these persons are usually due to multiple gestations and blood transfusions.

Our patient was a multipara and had no transfusion history in the past. The blood which caused to transfusion reaction was her own daughters' blood.

The immunological diagnosis of TRALI is important in order to prevent transfusions of donor blood containing HLA specific or neutrophil specific antibodies. Immunological diagnosis should be made by detecting granulocyte antibodies via immunofluorescence or agglutination methods. The classification of detected antibodies may be made by immunoblotting or immunoprecipitation antigen capture assay method. We could not manage to make a classification due to lacking of advanced laboratory

possibilities.

The treatment of TRALI is based upon a good respiratory care. Mechanical ventilation is not necessary in 70% of the cases. 30% O_2 with mask may usually be sufficient. Mortality may occur in 5% of cases because of the lung edema and pneumonia. Improvement should occur without sequela with reasonable treatment within 48-96 hours.

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References

1. Nishimura M, Ishikawa Y, Satake M. Activation of polymorphonuclear neutrophils by immune complex: possible involvement in development of transfusion-related acute lung injury. *Transfus Med.* 2004 Oct;14(5):359-67.
2. Kopko PM. Leukocyte Antibodies and Biologically Active Mediators in the Pathogenesis of Transfusion-related Acute Lung Injury. *Curr Hematol Rep.* 2004 Nov;3(6):456-61.
3. Han KS, Um TH. Frequency of neutrophil-specific antigens among Koreans using the granulocyte indirect immunofluorescence test (GIFT). *Immunohematol.* 1997 Mar;13(1):15-6.
4. Kopko PM. Review: transfusion-related acute lung injury: pathophysiology, laboratory investigation, and donor management. *Immunohematol.* 2004; 20(2):103-11
5. Popovsky MA, Haley NR. Further characterization of transfusion-related acute lung injury: demographics, clinical and laboratory features, and morbidity. *Immunohematol.* 2001 Dec;17(4):157-9.
6. Davoren A, Smith OP, Barnes CA, Lawlor E, Evans RG, Lucas GF. Case report: four donors with granulocyte-specific or HLA class I antibodies implicated in a case of transfusion-related acute lung injury (TRALI). *Immunohematol.* 2001 Dec;17(4):117-21.
7. Trzaska M, Pierzchala W. Transfusion related acute lung injury (TRALI)--case report. *Wiad Lek.* 2003;56(9-10):493-5.
8. Sachs UJ, Bux J. TRALI after the transfusion of cross-match-positive granulocytes. *Transfusion.* 2003 Dec;43(12):1683-6.
9. Palfi M, Berg S, Ernerudh J, Berlin G. A randomized controlled trial of transfusion-related acute lung injury: is plasma from multiparous blood donors dangerous? *Transfusion.* 2001 Mar;41(3):317-22.
10. Popovsky MA, Moore SB. Diagnostic and pathogenetic considerations in transfusion-related acute lung injury. *Transfusion.* 1985 Nov-Dec;25(6):573-7.
11. Weber JG, Warner MA. What is the incidence of perioperative transfusion-related acute lung injury? *Anesthesiology.* 1995;82:789
12. Popovsky MA, Abel MD, Moore SB. Transfusion-related acute lung injury associated with passive transfer of antileukocyte antibodies. *Am Rev.Respir.Dis.* 1983;128:1854
13. Florell SR, Velasco SE, Fine PG. Perioperative Recognition, Management, and Pathologic Diagnosis of Transfusion-related Acute Lung Injury. *Anesthesiology.*

1994;81:508-510

14. Keifer JC, Kingsley CP, Roth MT, Abt AB, Romano PJ. Transfusion-related Acute Lung Injury (TRALI) Complicating Colectomy for Ulcerative Colitis.

Anesthesiology. 1998; 89:1020-3.

15. Warner MA, Weber JG, Warner ME: Etiologies and incidence of acute pulmonary edema in the immediate perioperative period. *Anesth Analg.* 1990;70: 421.

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