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TRANSFUSION-RELATED ACUTE LUNG INJURY (TRALI) AFTER DELIVERY - A CASE REPORT

Dilek UYGUR[1], M.D., Ömer Lütfi TAPISIZ[1], M.D., Behiye AKKALYONCU[2], M.D., Mustafa UGUR[1], M.D.,

TRALI is an underdiagnosed serious complication of blood transfusion characterized by the rapid onset of respiratory distress, hypoxia, and non-cardiogenic pulmonary edema during or soon after blood transfusion. We report a case of TRALI caused by transfusion of whole blood. The 24-year-old patient had postpartum hemorrhage 23 hours after vaginal delivery. The patient received 2 units of whole blood. She developed acute respiratory distress syndrome immediately after the transfusion. Resolution of symptoms occurred in 5 days with appropriate ventilatory support and fluid management.

Key Words: Respiratory distress, transfusion reaction, TRALI, leucocyte antibody.

DOĞUM SONRASI TRANSFUZYONA BAĞLI AKUT AKCİĞER HASARI (TRALI) - OLGU SUNUMU

TRALI, kan transfüzyonunun çoğu zaman klinisyenler tarafından tanı konulamayan ancak oldukça ciddi bir komplikasyonudur. Kan transfüzyonundan hemen sonra gelişen respiratuar distres, hipoksi ve kardiyojenik olmayan pulmoner ödem ile karakterizedir. Postpartum 23. saatte vaginal kanama nedeniyle 2 ünite tam kan verilen hastada kan transfüzyonu bitiminde dispne gelişti. Klinik olarak TRALI ile uyumlu olduğu belirlenen hasta uygun oksijen ve sıvı desteği ile tedavi edildi.

Anahtar Sözcükler: Respiratuvar distres, transfüzyon reaksiyonu, lökosit antikoru.

Zekai Tahir Burak Women Health Care, Research and Education Hospital[1] Ankara, Turkey
Atatürk Chest Disease and Thoracic Surgery Hospital[2] Ankara, Turkey

INTRODUCTION

Transfusion-related acute lung injury (TRALI) is a rare but life-threatening complication of transfusion (1). TRALI is clinically similar to adult respiratory distress syndrome (ARDS) but has a much better prognosis (2). A mortality rate of 5 to 10 percent has been reported, as compared with a rate of 50 to 60 percent for ARDS (2). The reaction usually starts within 6 hours of transfusion and is characterized by severe pulmonary edema, severe hypoxemia, hypotension, and chills and fever (1). Cardiogenic and other causes of respiratory distress should be excluded. In most cases, TRALI improves clinically within 48-96 hours of onset.

The pathogenesis of TRALI is not fully understood, but it is commonly associated with the transfusion of donor plasma containing white blood cell (WBC) antibodies. Recently, it has been suggested that biologically active lipids can also cause TRALI (3). We describe a case that was clinically compatible with TRALI.

CASE REPORT

A 24-year-old woman, gravida 1, para 0, was admitted to our obstetric department at 39 weeks of gestation, with ruptured membranes and cephalic presentation. Prepartum blood count results were as follows: hemoglobin (Hb), 13.1 g/dl; WBC, 12,500/µl; and platelets, 206 x 10^9/l. The patient delivered vaginally without any complication. After 23 hours, it was noticed that the patient had vaginal bleeding, which was thought to originate from the episiotomy site. The patient was transferred to the operating room and the episiotomy and some vaginal lacerations were repaired. The patient's Hb was 9.1 g/dl and the WBC count was 18,500/µl. Since the Hb level of the patient had decreased from 13.1 to 9.1 g/dl, 2 units of whole blood were transfused. The patient became dyspneic and agitated immediately after the transfusion. Her physical examination revealed widespread bilateral pulmonary rales, but no cardiac murmur. Her blood pressure was 80/50 mmHg. She did not have a fever. There was no clinical evidence of cardiac failure, sepsis, or chest infection. The chest X-ray showed bilateral pulmonary infiltrates and a normal cardiac outline, which were consistent with TRALI (Fig. 1). Pulse oximetry hemoglobin oxygen saturation was recorded as 80% despite an oxygen fraction of 100%. A contrast enhanced computerized tomography (CT) scan on post-transfusion day two showed dense infiltrations and air bronchograms in especially the upper and medium lung lobes, consistent with focal air space consolidation (Fig. 2). Despite the dramatic pulmonary changes on the CT scan, the patient's residual respiratory function was assessed clinically as sufficient to delay the initiation of mechanical ventilation. Management included intensive respiratory support. Respiratory function improved gradually and the patient was transferred to a routine care unit on the 5th day. A repeat X-ray on that day showed clearing of the pulmonary shadow (Fig. 3).

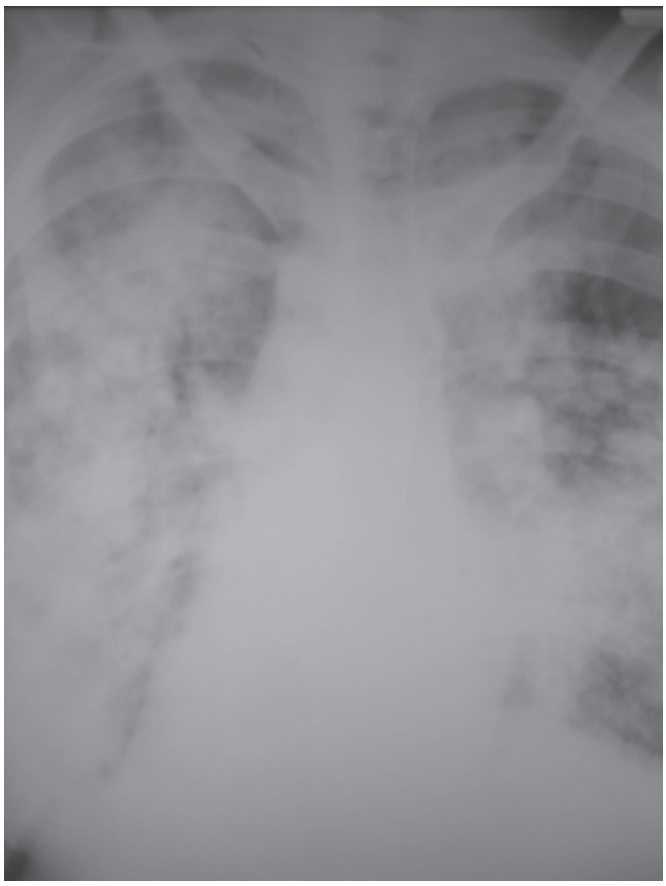

Figure 1: Post-transfusion chest radiograph consistent with bilateral pulmonary edema.

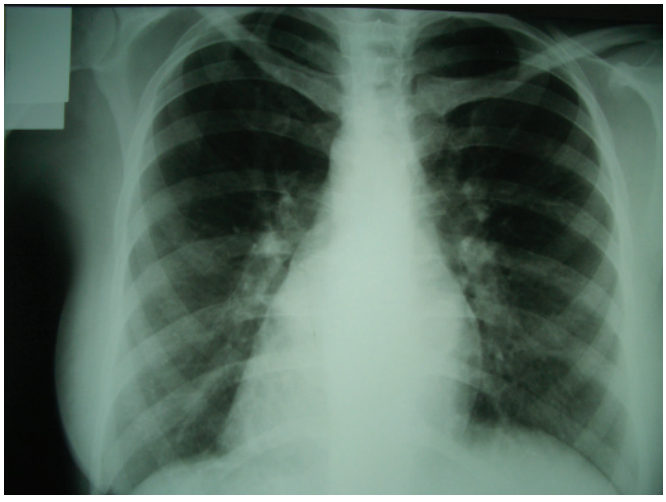

Figure 3: Repeat X-ray 5 days after transfusion demonstrating clearance of the pulmonary shadow.

DISCUSSION

TRALI is almost certainly under-diagnosed. Many clinicians are unaware of the condition or may not recognize transfusion as the cause when it occurs. TRALI describes a particular form of ARDS that occurs after transfusion and which is caused by antibodies in plasma of a single donor unit reacting with leucocyte antigens in the recipient.

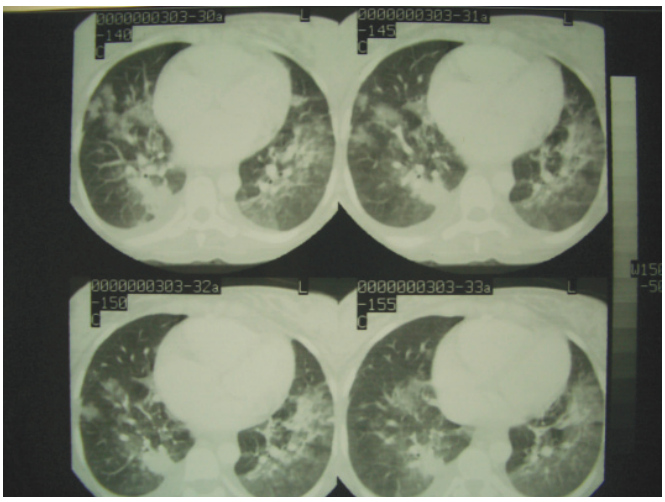

Figure 2: Computerized tomography scan of the chest, two days after transfusion, demonstrating dense infiltration and air bronchograms consistent with air space consolidation.

The condition is characterized by the sudden onset of non-cardiogenic pulmonary edema, often with marked systemic hypovolaemia and hypotension, occurring during or within a few hours of transfusion (1). Fever and rigours are reported, but may be absent or relatively mild. There is rapid onset of severe hypoxia, a chest radiograph typical of ARDS, and copious frothy yellow or pink fluid in the trachea. Diagnosis of TRALI depends on the exclusion of other causes of pulmonary edema or ARDS. Laboratory findings may include unexpected haemoconcentration and a sudden fall in serum albumin (4). The diagnosis of TRALI may be aided by screening donor plasma for granulocyte or human leukocyte antigen (HLA) antibodies. However, limited availability of testing, the time taken to obtain the screen, as well as reported cases of TRALI occurring without a positive antibody screen, mean that TRALI is predominantly a clinical diagnosis.

The key to distinguish TRALI from other forms of pulmonary edema is recognition that the pulmonary edema is non-cardiogenic and that affected patients do not have volume overload. This distinction is important because treating patients with TRALI with aggressive diuresis can result in further hypotension, shock, and death. Treatment should consist of maintenance of hemodynamic status and ventilatory assistance. Steroids in high doses have been used, partly on the assumption of an immunological cause, but there is no direct evidence of benefit when they are given after the initial insult (5). Measurement of both the central venous pressure and pulmonary artery wedge pressure may be helpful, both for diagnosis and the best fluid management, and clinical improvement has been seen after restoration of circulating volume with saline or albumin (5).

TRALI has been estimated to occur about once in 5000 transfusions (6). The precise mechanism of TRALI is unknown, but it may be an immune-mediated event. Donor antibodies, rather than recipient antibodies as in other transfusion reactions, may be the pathological trigger (1). Granulocyte or

HLA antibodies corresponding to recipient epitopes have been implicated in various studies. Infusion of donor plasma containing antibodies may activate recipient white cells to produce inflammatory mediators, leading to increased vascular permeability. Direct targeting of host pulmonary vascular endothelium by transfused donor antibodies is an additional hypothesis. Multiparous women may have an increased incidence of foreign white cell antibodies, thought to be due to exposure to fetal white cell antigens during pregnancy. Transfusion of plasma from multiparous donors may lead to a higher incidence of recipient TRALI (6). Recently, Silliman et al. proposed an alternative, "two-hit" hypothesis to explain the pathogenesis of TRALI. The first hit is the patient's pre-existing condition (i.e. infection, cytokine administration, recent surgery, or massive transfusion), and the second hit is the infusion of biologically active lipids in stored blood components (3). Thus a patient may develop TRALI after receiving blood components that do not contain WBC antibodies.

The present report emphasizes that the clinician should carefully balance the benefits and risks of transfusion. TRALI should be included in the differential diagnosis of respiratory distress (with or without pulmonary edema or ARDS) in the setting of blood and component transfusions. Proper awareness of TRALI will lead to a prompt diagnosis and appropriate management.

Correspondence Address
Dr. Omer Lutfi TAPISIZ
Turan Gunes Bulvari, Sedir Sitesi
C2 Blok No: 6, Or-an, 06450 Ankara, TURKEY
Tel: 0 312 4917707
Fax:0 312 4260004
E-mail: omertapisiz@yahoo.com.tr

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