SPECIAL SECTION: OBSTETRICS & GYNAECOLOGY

TRANSFUSION RELATED ACUTE LUNG INJURY IN MULTIPAROUS WOMEN: TWO CASE SCENARIOS

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Abstract

Transfusion related acute lung injury (TRALI) is defined as acute lung injury occurring during or within 6 hours of transfusion with clear temporal relationship to transfusion. An immune mediated mechanism has been implicated.

We report two cases of TRALI. The subject in Case 1 underwent laparotomy, received 7 units of blood products and developed features of TRALI. Case 2 was given transfusion for preoperative correction of anaemia and developed TRALI.

Both women were multiparous with one receiving multiple units of blood products. The gender of donors could not be traced and the other developing TRALI with one unit of blood, the donor being a male with no previous history of transfusion. As the donors are usually males, the chance of antibodies against WBCs in the recipient acting against the recipients’ antigens is high. Hence
TRALI is life threatening, diagnosed only with a high index of suspicion, especially in multiparous women with symptoms of TRALI.

Introduction

The term TRALI – transfusion related acute lung injury was coined by Popovsky et al in the early 1980’s and described the relationship to luecoagglutinins in the blood component. The National Heart, Lung, and Blood Institute [NHLBI] defined TRALI as acute lung injury occurring during or within 6 hours after a transfusion, with a clear temporal relationship to transfusion in patients without or with risk factors for ALI, other than transfusion. Limitations to the definition of NHLBI was that it did not include cases with pre-existing ALI, onset more than 6 hours, lack of lab diagnostic criteria and a severity of hypoxia that would miss subtle cases of TRALI. Canadian Consensus Conference modified the criteria of TRALI. The criteria included acute lung injury- acute onset with bilateral infiltrates on frontal chest radiograph, no evidence of left arterial hypertension, no pre-existing ALI before transfusion, no temporal relationship to an alternative risk factor for ALI and features of hypoxemia: ratio of PaO2/FiO2 < or = 300 or SpO2 < 90 % on room air and other evidence of hypoxia in non research setting. Even though the exact mechanism of TRALI is not well understood, an immune mediated mechanism has been implicated in most cases. We are reporting two cases of TRALI not because it is infrequent but to create awareness among the medical fraternity about this life threatening condition which is undiagnosed and under estimated.

Case report 1

A 40 year old female, para 3, live 3, with full term normal deliveries, was admitted to a private hospital as a case of ruptured ectopic in shock. Emergency laparotomy was done and the diagnosis was confirmed. Peritoneal cavity contained 2 litres of blood. Right tube isthmic region was the sight of ectopic and a total salpingectomy was done. She was resuscitated with 5 units of packed cells, 1 unit of FFP and 1 unit of cryoprecipitate. After 6-8 hours of surgery, she developed dyspnoea with spo2 falling to 70. She was referred to a tertiary care centre with a diagnosis of respiratory distress with possibility of TRALI. At the time of examination in the casualty, patient was conscious, oriented and with a blood pressure of 90/60 mm Hg, pulse rate 70 /minute, SPO2 98% with oxygen and a
respiratory rate of 40/minute, Air entry in the chest was reduced with bilateral rhonchi and crepitations. Abdomen was soft with mild gaseous distension. Chest X-ray showed bilateral diffuse opacities. ABG showed partly compensated respiratory acidosis with low PO2. Later patient developed tachycardia, hypotension, fever, with SPO2 falling to 85% with respiratory rate of 50/minute in spite of oxygen therapy. Patient was put on ventilatory support and was given broad spectrum antibiotics and methyl prednisolone. The patient became stabilised the next day after intubation and was extubated on the 3rd day. Patient improved with nebulisation, antibiotics, chest physiotherapy and low molecular weight heparin and was discharged without any sequale.

Case report 2

A 50 years old female para 3, live 3 with full term deliveries with last child birth 25 years back, was admitted to a private hospital with ovarian tumor. She required pre-operative transfusion for correction of anaemia. She had no previous transfusions. One unit of A+ve compatible packed cell transfusion was given with continuous monitoring of SPO2, blood pressure and pulse rate. Even though the transfusion period was uneventful, patient developed mild shivering and giddiness, five hours after transfusion with oxygen saturation falling down to 85%. Immediately she was supported with steroids and oxygen. But she soon developed bradycardia, hypoxemia and hypotension with scattered crepitations in the chest. There was no evidence of haemoglobinuria.O2 saturation and blood pressure were maintained with oxygen, dopamine and steroids for 48 hours and patient was weaned of steroids slowly. On the 4th day, patient became symptomatically better. She was discharged and shifted to a tertiary centre for surgical management. Staging laparotomy was done later. She was transfused with leucoreduced red blood cells in the peri-operative period and did not develop any reaction. The postoperative period was uneventful.

In both cases, transfusion associated circulatory overload (TACO) was ruled out by normal ECG and blood compatibility.

Discussion

TRALI has a clinical presentation similar to acute respiratory distress syndrome (ARDS) occurring in the setting of transfusion. Patients present with dyspnoea, hypoxia, pulmonary oedema and bilateral fluffy infiltrates on chest radiograph during or within 6 hours of transfusion, with majority of cases occurring within 1-2 hours of transfusion.² Signs and symptoms include tachypnoea, frothy pulmonary secretions, hypotension [less commonly hypertension], fever, tachycardia and cyanosis with auscultation of lung fields showing diffuse
rales. Case -1 had all features of TRALI such as tachycardia, fever, hypotension, dyspnoea with rales on auscultation, and opacities on X-ray chest, and improved with ventilatory support. In Case 2, patient developed hypotension, dyspnoea and chest signs which improved with oxygen. Both showed typical features of TRALI.

There are no specific investigations for TRALI. Laboratory investigations like leucopenia, acute transient neutropenia, monocytopenia, presence of matching leukocyte antigen - antibody in donor, recipient, increased neutrophil priming activity in transfused blood and hypocomplementemia are of limited value as they are only suggestive and not diagnostic of TRALI.4

Differential diagnosis include TACO, anaphylactoid reaction to transfusion, bacterial contamination of transfused component and haemolytic transfusion reaction.

An antibody mediated mechanism has been postulated in most cases of TRALI. It is postulated that passive transfer of leucoagglutinating antibodies via transfusion of plasma containing blood components results in binding to recipient neutrophil and these antibody bound neutrophils get activated and sequestered in the lungs where complement activation and release of neutrophil bioactive products results in endothelial damage, capillary leak and ALI. In 65-90% of reported cases of TRALI, leukocyte antibodies have been identified in the donor and the corresponding antigen was identified in the recipient neutrophils. 2

Antibodies in TRALI include HLA class 1, 11 and neutrophil specific antibodies.2,4,12-16 In a small percent of cases, the leucoagglutinating antibodies appear to be from the recipient and are directed at transfused neutrophils.17,19 Most of the donors implicated in TRALI have been multiparous women who became alloimmunised during pregnancy. The prevalence of HLA class 1 and 11 antibodies is highly correlated with parity, with alloimmunisation rates of 1.7 %, 11.2 %, 22.3%, and 29.8% in women with 0, 1, 2, 3 or more pregnancies respectively.20 In our country, most of the donors are males, and female donors are less than 2 % [personal communication]. In both the cases reported here, the recipients were multiparous with previous three deliveries and might have become alloimmunised through pregnancy. So the probability could be that the antibodies of the recipients could have bound to the transfused neutrophil causing ALI.

Treatment of TRALI is mainly supportive with oxygen supplementation and in most cases with ventilatory support. In contrast to other cases of ARDS, patients with TRALI recover quickly, with resolution of pulmonary infiltrates within 96 hours of
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Transfusion. The mortality rate is 5-10%. In both of our cases, patients improved dramatically on ventilatory support and with oxygen supplementation. Both patients recovered within 96 hours.

TRALI has emerged as the leading cause of transfusion related mortality reported to the FDA since 2003. In 2006, there were 35 reported deaths due to TRALI, more than all other causes combined. The true incidence of TRALI is unknown because a standard definition has only been recently developed. TRALI has been reported in all types of blood components and most implicated blood products contain less than 50 ml of plasma. Fresh frozen plasma has been implicated most frequently in TRALI cases and, TRALI related deaths reported to FDA and United Kingdom. In the case 1, red blood cells, fresh frozen plasma and cryoprecipitate had been transfused and any of these components could have caused the reaction but in case 2, reaction developed after transfusing 1 unit of packed cells. Hence it is probable that the second patient may have antibodies against neutrophils in the red blood cell unit.

In our country, the number of TRALI cases is greatly under reported. The published incidence of TRALI ranges from 0.02 % to 0.05 % per blood unit transfused and from 0.08% to 0.16 % per patient receiving transfusion. Recurrent TRALI has been reported and hence it is important to be careful before giving any further transfusions to a patient who had an episode of TRALI.

Conclusions

TRALI is a life threatening situation, the diagnosis of which requires high index of suspicion. The diagnosis is mainly based on exclusion of other causes. Correct diagnosis is important because diuretics are contraindicated and hypovolemia needs to be corrected in TRALI. Multiparous female donors are considered to be at higher risk. In Indian population, very few females donate, are parous and have the chance of antibodies in the recipient, acting against donor antigen. Therefore it is important to consider TRALI as a differential diagnosis in patients with respiratory distress in a transfusion setting, especially in female patients.

References


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related acute lung injury.


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