Case Report

TRANSFUSION RELATED ACUTE LUNG INJURY: A case report

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INTRODUCTION

Acute pulmonary edema has been reported following whole blood transfusions or its components in the medical literature.[1-3] Transfusion Related Acute Lung Injury(TRALI) is a life threatening complication of blood and its components resembling Acute Respioratory Distress Syndrome (ARDS) or ALI (Acute Lung Injury). It was first reported in 1992.[4] TRALI is characterized by dyspnoea, hypoxemia, hypotension, bilateral pulmonary edema and fever despite proper crossmatching of blood.[2-6] TRALI has been linked to the presence of granulocyte antibodies, HLA class I & II antibodies and biologically active lipids in donor plasma.[6-8] TRALI has been estimated to occur within six hours of transfusion of blood or its components or IVIg.[8,9,10,12.]

CASE REPORT

Twenty nine year old philipino house wife (2nd gravida) had a normal full term vaginal delivery with right mediolateral episiotomy, She had postpartum heamorrhage due to vaginal lacerations, she was observed in the delivery room and she continued to bleed then she was taken to Observation Room(OR) for examination sedated with midazolam +ketamine analgesia, but on examination she continued to bleed from raw area of cervix and from sutured lacerations and episiotomy.The patient was started on a crossmatched whole blood transfusion but after the first unit she complained of dyspnoea and Tachypnoea was noted by the anaesthesiologist and he shifted her to the Intensive care unit where she developed frank pulmonary edema when she was started on 3rd unit of whole blood, She had bilateral basal crackles and her SpO_2 was 72%, She was given intravenous Furosemide 40 mg and and Internist was called, The 3rd blood transfusion was in progress, it was stopped immediately and blood was sent for recheck with the blood bank for reassessment and re-cross matching which was found to be compatible with the patient.

Her arterial blood gases showed hypxemia(pH 7.35, pO_2 84%, pCO_2 25, HCO_3 13.4 and BE -10 and Her BP was also low 100/70 and respiratory rate of 35/ minute and patient was cyanosed and her ECG showed sinus tachycardia and her portable Chest x-ray showed interstitial shadowing in both prehilar regions and bilateral basal zones, At this juncture it was decided to intubate her. An intubation on froth and fluid came out of laryngeal opening. The patient was put on intravenous Dextrose saline with 20meq KCl @ 60ml/hour, intravenous Methyprednisolone, intravenous ceftriaxone and pulmicort and salbutamol nebulizations, The patient was on assisted ventilation for 24 hours and she was extubated after 24 hours when her arterial blood gases improved, She made an uneventful recovery and steroids were stopped on fifth day with out any untoward incident, Her Blood Urea and Serum Creatinine were within normal limits and screening test for ANA (antinuclear antibody) Anti-dsDNA, AntiSm were

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negative. We suspected TRALI versus ARDS or overtransfusion because only one litre of Ringer was transfused and this is not significant amount in an otherwise healthy patient. There was no previous history of chest infection and patient never had history of valvular heart disease or lung disease in the past and her first delivery was uneventful. Her echocardiographic examination was within normal limits.

CBC and platelet counts were within normal limits, her initial prothrombin time was 17 seconds (Control 13s) and partial thromboplastin time was 56 seconds (control 45 sec.) and bleeding time was 3 minutes (<7min) and her Fibringen level was 200mg/dl (within normal limits) and FDP (Fibrin degradation products) were within normal limits. Her PTT and PT improved within few days without specific treatment.

DISCUSSION

The cases of TRALI have been reported in the past but the entity has very recently been recognized and reported.[6-10] Usually the donors implicated in TRALI cases are multiparous females but it has been reported after male blood donors like in our case it was male donors and had the same ethnic background as the patient and no antigranulocyte antibodies were found in the donors sera. Chromosomal analysis was not done. Although the pathogenesis of TRALI is unknown there is sufficient clue to implicate an immune reaction and unlike most immunologically mediated transfusion reactions the pathologic autoantibodies in TRALI originate from donors rather than recipients. Antibodies in the donor are suspected to be the causative agent because the "substrate" is the entire circulatory system and marginated pool of leucocytes. Since there is no diagnostic test or a pathognomonic sign of TRALI , it is a diagnosis of exclusion; one must rule out other causes of respiratory distress and pulmonary edema in patients receiving transfusions, Myocardial infarctions, Circulatory overload and bacterial infections and any other cause of ARDS specially SARS. Normal CVP is consistent with the diagnosis of TRALI. The treatment of TRALI is symptomatic, pressor agents for sustained hypotension. Corticosteroids are of marginal value and diuretics have no role because it is a microvascular injury rather than fluid overload. The question why the lung is the primary endorgan of choice in TRALI is not answered.[5] It is recommended that if a TRALI case occurs the donor should be temporarily suspended for three moths and 14 ml of clotted and 7ml EDTA blood obtained from the donor, fresh samples of whole blood should be obtained (plasma is not appropriate) and sent for HLA type I & II antibodies[5]. TRALI incidence is much higher than is thought of and is still an underdiagnosed and under reported illness.[11,12]

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