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Case Report

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Transfusion related acute lung injury (TRALI): When and how much to transfuse?

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ABSTRACT

In a patient with reduced blood profile (reduced platelet count , decreased haemoglobin, reduced clotting factor or dearranged PT INR), it is a common practice to transfuse the patients with Blood products. Clinicians normally overdo the transfusion and neglect the negative outcome of transfusion reactions, which can be life threatning and very difficult to manage..Here we are presenting a case of thrombocytopenia who was transfused with RDP'S (random donar platelet) and later developed TRALI,a potentially fatal complication of blood product transfusion in which a patient develops rapid onset lung injury resulting in noncardiogenic pulmonary edema due to activation of immune cells in the lungs. He was further managed with mechanical ventilation and symptomatic treatment. One should exercise restrain while transfusing and transfuse only when there is a definitive indication and always watch the patient for development of respiratory distress due to TRALI. Despite aggressive support, mortality of more than 12% are reported once TRALI is diagnosed. The risk and benefit of all blood products should be assessed before transfusion.

Key Words: *Trali Taco Lung Injury Pulmonary Edema Transfusion Reactions*

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INTRODUCTION

Transfusion-related acute lung injury (TRALI) is a serious and potentially fatal complication of blood product transfusion in which a patient develops rapid onset lung injury resulting innoncardiogenic pulmonary edema due to activation of immune cells in the lungs. It is one of the leading cause of transfusion related morbidity and mortality. It is more common in critically ill patients in the Intensive Care Unit. Now a days blood and blood product transfusion has become common due to dengue fever and easy availability. As a result, it is important for the physicians to take a judicious decision about transfusion of blood and blood products considering all pro and con.

Case Details

55-year-old male patient presented in emergency dept (ED)with history of fever, abdominal pain, and weakness. He was a known case of type II diabetes mellitus but not on proper medication. He was an occasional smoker and drinker. There was no other significant past history. Vitals on presentation was BP - 130/68 mm Hg, pulse – 107/min, SpO2 -97%, Breath rate -19 /min and random blood sugar in emergency dept(ED) 282 mg/dl. Routine CBC revealed Platelet count 9000/mm^3. His chest x ray taken in emergency dept on admission is shown in Fig 1.

Patient's treatment started in ED with insulin, antibiotics, and antipyretics. He was transfused with 4 Random donor platelets (RDP) for thrombocytopenia. However, within few hours of platelets transfusion, patient developed difficulty in breathing and SpO_2 dropped to 85%. He was initially managed with face mask oxygen and Non-invasive ventilation. Repeat Chest radiograph done next day revealed multiple bilateral non homogenous opacities suggestive of pulmonary edema Fig 2. HRCT chest done next day showed bilateral multiple ground glass opacities suggestive of pulmonary edema Fig 3.

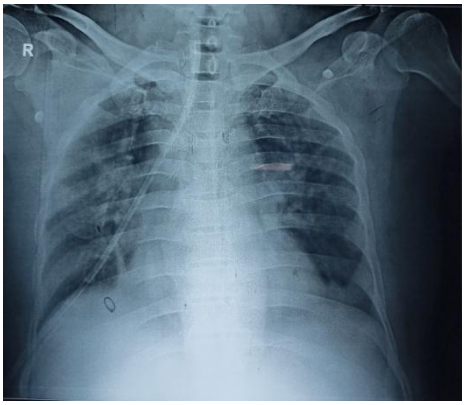

Fig 1: Chest X Ray on admission.

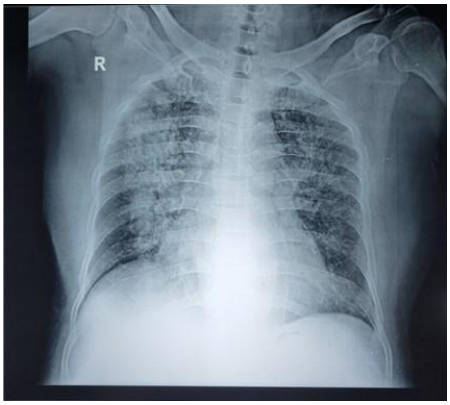

Fig 2: Chest X Ray showing bilateral non Homogenous opacities

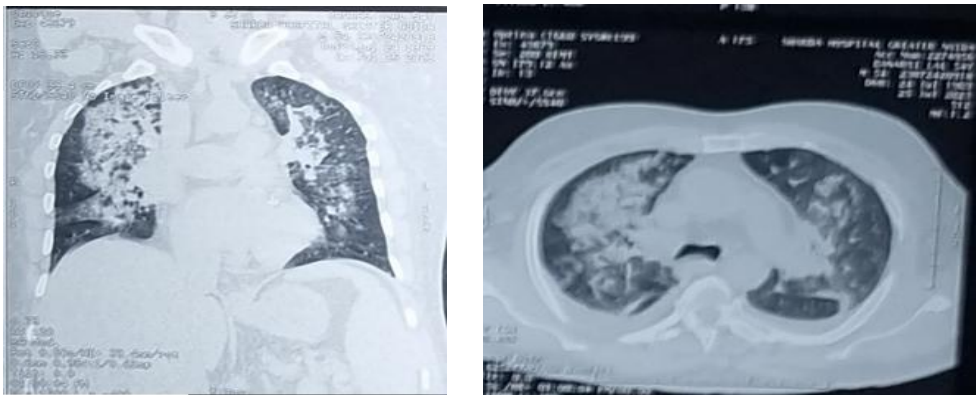

Fig 3:HRCT chest showing bilateral multiple ground glass opacities suggestive of pulmonary edema

Patient was shifted to intensive care unit (ICU) for further management. Shortly after shifting to ICU, his clinical condition further deteriorated and he developed obvious respiratory distress with tachypnoea and tachycardia. It was decided to initiate invasive mechanical ventilation, so his trachea was intubated and was placed on volume control mode of ventilator withFi02 50%, TV-450ml, RR-20, PEEP-8. He developed hypertension for which first he was started on labetalol and nitroglycerine infusion. After about 03 days of mechanical ventilation patient improved clinically, Chest X ray showed clearance of opacities, Arterial blood gas showed correction of blood gas abnormalities. Patient was given short weaning trial following which he was successfully extubated. Vitals post extubation were BP- 159/91 mm Hg, Pulse – 84/min, SpO2 – 94%, breath rate-20/min. Post extubation he was placed on intermittent NIV for 24 hour. There was further improvement in his condition with NIV and he was transferred to medicine ward.

DISCUSSION

Diagnostic criteria for TRALI have been recently developed and primarily consists of hypoxia and bilateral pulmonary edema occurring during or within 6 hours of a transfusion in the absence of cardiac failure or intravascular volume overload. All types of blood products have been associated with TRALI, however the plasma rich components such as fresh frozen plasma and apheresis platelets, have been most frequently implicated [1].

The pathogenesis of TRALI is not completely understood. Leukocyte antibodies in donor plasma have been implicated in most cases with antibodies directed at human leukocyte antigen (HLA) class I and HLA class II or neutrophil-specific antigens, particularlyHNA-3a.Activation of pulmonary endothelium is important in development of TRALI. Transfused leukoagglutinating antibodies bind to recipients neutrophils localized to pulmonary endothelium resulting in activation and release of oxidases and other damaging biological response modifiers that causes capillary leak [2].

It has, however, recently been recognized that the transfusion of blood products in critically ill or injured patients increases the risk (odds ratio 2.13; 95% confidence interval 1.75-2.52) for the development of the acute lung injury 6-72 hours after the transfusion. This "delayed TRALI syndrome" is common, occurring in up to 25% of critically ill patients receiving a blood transfusion, and is associated with a mortality of up to 40%. While the delayed TRALI syndrome can develop after the transfusion of a single unit, the risk increases as the number of transfused blood products increase. The management of both the classic and delayed TRALI syndromes is essentially supportive [3].

Immediate management of TRALI is to stop the transfusion and notify blood bank to screen the donar unit for antileukocyte antibodies, anti HLA or antineutrophil specific antibodies. Treatment consists of oxygen support, non-invasive and invasive mechanical ventilation. Diuresis is not indicated, and the role of steroid is unproven. Patients typically recovers within few days. Best practice is prevention. In UK the incidence of TRALI was substantially reduced by using plasma from male donor and screening female donor for HLA and HNA antibodies which are strong risk factors. It is important to keep the possibility of transfusion associated circulatory overload (TACO) as a differential diagnosis of TRALI, however the differentiation can be difficult. TACO and TRALI have emerged as important causes of post transfusion morbidity and mortality. As understanding of their pathogenesis improves, incidence, risk factors, differences, and possible preventive interventions are becoming clearer. There is no sentinel feature that distinguishes TRALI from TACO. Developing a thorough clinical profile including presenting signs and symptoms, fluid status, cardiac status including measurement of brain natriuretic peptide, and leukocyte antibody testing is the best strategy currently available to distinguish the two disorders [4].

In our Institute somewhat similar clinical picture developed in 02 more patients with severe dengue while receiving platelets transfusion. One should exercise restrain while transfusing platelets to dengue patients, transfuse only when there is a definitive indication and always watch the patient for development of respiratory distress due to TRALI. Despite aggressive support, mortality of more than 12% are reported once TRALI is diagnosed. Even those who survive has a slow recovery. The risk and benefit of all blood products should be assessed before transfusion. Blood centers are implementing male predominant plasma programs to limit TRALI, and preliminary evidence suggests that this is a useful intervention.

Is it possible to predict the development of TRALI following transfusion of blood products? HLA antigens can be matched when administering blood products. Research has demonstrated that blood banks that use the HLA genotypes of the blood donors have better transfusion outcomes. There has been a discussion of the possibility of creating a database with genotyped donors to make it slightly easier to find blood for patients with rare HLA antibodies so that their transfusions can have less risk of adverse outcomes because of having matched donors [5].

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