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CASE REPORT

RARE BLOOD TRANSFUSION HAZARD-TRALI: A CASE REPORT

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ABSTRACT

Transfusion related acute lung injury is a rare life-threatening complication of blood transfusion. The diagnosis is done clinically as there are no distinguishing biomarkers. We report a case of 25 years old female with six weeks of gestation with ruptured ectopic, who presented to the emergency department with acute abdominal pain. She underwent emergency laparotomy and received two units of blood transfusion post-operatively. Eight hours after surgery and six hours following blood transfusion, the patient developed fever (101^0 F), tachycardia (> 140 bpm) and respiratory distress (SPO2: <80%, Respiratory rate: >30 per minute). The diagnosis of transfusion related acute lung injury was made and managed accordingly in Intensive Care Unit. Being a potentially life-threatening complication of the blood transfusion, early clinical diagnosis and further management is crucial to prevent fatal outcome.

INTRODUCTION

Transfusion-related acute lung injury (TRALI) is a rare but often devastating complication of blood transfusion. TRALI is the third most common cause of fatal transfusion reactions next to ABO blood type incompatibility and hepatitis.[1] The incidence of TRALI has been reported to be about 0.01 to 0.08 % per plasma containing unit transfused and has an associated mortality rate of 5 - 14 %.[1,2] As distinguishing biomarkers are absent, TRALI is a clinical diagnosis. The lack of a standardized definition of TRALI has contributed to under-diagnosing of this syndrome and has also hindered its epidemiology and research investigation.[3] We present a case of TRALI that was diagnosed and managed at our center considering the rarity of case and importance of need for early recognition and prompt management.

CASE REPORT

A 25-year-old second gravida, parity one with previous lower segment caesarian section presented to emergency department of Manipal Teaching Hospital (MTH) with complain of amenorrhea for six weeks and abdominal pain. Pain was located over left iliac fossa that radiated to suprapubic region and also had per vaginal spotting for a day. She had no history of dizziness, loss of consciousness, shoulder pain, fever. On examination she was pale but hemodynamically stable. Tenderness was present over suprapubic region. Per speculum cervical examination showed minimal bleeding. On per vaginal examination, uterus was normal size with tenderness over left adnexa and cervical motion tenderness was present. Her urine beta hCG was positive. Ultrasonography revealed heterogeneous hypoechoic mass in left adnexa and pouch of Douglas with moderate free fluid in peritoneum suggesting ruptured ectopic pregnancy. Her hemoglobin was 6 gm/dl.

Emergency laparotomy was performed with hemodynamic resuscitation of the patient. There was gross hemoperitoneum with 2000 ml of blood in the peritoneal cavity. There was left ampullary abortion; uterus, left and right ovary and right tube was normal. Left salpingectomy was done followed by peritoneal washing. Blood transfusion was started as soon as the blood was made available. Eight hours post operatively and six hours following second unit of blood transfusion, the patient developed tachypnea (respiratory rate was > 30/min), tachycardia (heart rate > 140/minute), fever (101°F), decreased SPO2 (<80%). She also developed icterus and crepitation in bilateral chest. She was shifted to Intensive Care Unit (ICU) of MTH with clinical diagnosis transfusion-related acute lung

injury. Complete blood count (CBC) revealed WBC count: 9,400 /mm^3 (Neutrophil: 78%, Lymphocyte: 20%), Hemoglobin: 6 gm/dl, Platelet count: 1, 52,000/ mm^3. Liver function test (LFT) was deranged (Total bilirubin: 19.1 mg/dl, direct: 6.3 mg/dl, AST: 22U/L, ALT: 23 U/L, Total protein: 5.2 gm/dl). Kidney function test was normal. Chest X- ray revealed bilateral haziness with alveolar and interstitial infiltrates. Arterial blood gas analysis (ABG) was done which showed: pH: 7.39. pCO_2: 32 mmHg, pO_2: 82 mmHg. FiO_2: 57%. PaO_2/FiO_2 was 136.6. Central venous line was opened. She was managed with IV Furosemide and Hydrocortisone. Her urine output was 80ml/hr in first 12 hours. The patient did not require intubation as she improved significantly with high flow oxygen via face mask that was started at 6-8L/minute. Over the next 12-14 hours she continued to improve; her respiratory rate settled down to 20 -25/min and she was able to maintain oxygen saturation of more than 95% oxygen at 3 L/minute. On 7th postoperative day her saturation was maintained on room air and was shifted to ward on 8th postoperative day. She improved over period of ten days and was discharged on 11th postoperative day. Prior to discharge, echocardiography was done, which revealed no evidence of any preexisting cardiac disease.

DISCUSSION

Transfusion related acute lung injury (TRALI) is a rare but life-threatening complication of blood transfusion which is being increasingly recognized. It presents with pulmonary insufficiency and radiography evidence of pulmonary edema with normal cardiac function.[4]

The pathophysiology is related to two predominant hypotheses– increased pulmonary capillary permeability induced by anti-leukocyte antibodies in the donor blood or neutrophil activation induced by biologically active agents like lipids and cytokines.[5] Anti-leukocyte antibodies activate the recipient leukocytes, leading to sequestration of the recipient leukocytes within the lung with subsequent degranulation, resulting in increased capillary permeability. The other mechanism is the activation of the pulmonary endothelium by underlying problems like sepsis, inflammation, etc., which leads to neutrophil sequestration within the lung and results in pulmonary capillary leak when donor antibodies activate these "primed" neutrophils.

The National Heart, Lung and Blood Institute proposed a definition of TRALI as Acute Lung Injury (ALI) occurring during or within six hours of a transfusion in patients without preexisting ALI before transfusion.[3] The diagnosis of TRALI should be considered in all cases of respiratory distress with significant hypoxemia with PaO2/FiO2 ratio less than 300mmHg, bilateral chest infiltrates on chest radiographs temporally related to transfusion, like our patient with PaO2/FiO2 ratio 136.6 mmHg and pulmonary infiltrates appear at the time of the reaction that resolved within 96 hours. Other TRALI related signs reported are fever, tachycardia, hypotension, cyanosis and transient leucopenia as seen in our case. Plasma containing blood components such as whole blood, aphaeretic platelet concentrates, fresh frozen plasma, packed red cells, granulocytes, cryoprecipitate and intravenous immunoglobulin have all been implicated in TRALI.[5]

Symptoms of TRALI generally develop within 6 hours of transfusion, most often occurring within first 2 hours after transfusion is started. In our case the clinical features of fever, hypoxia and respiratory distress developed in six hours following two units of whole blood transfusion. The presentation varies between mild pulmonary congestion to frank pulmonary edema and subsequent acute respiratory distress syndrome. Reduction in blood pressure is not a consistent finding but transient neutropenia has been described.[6] Our patient had pulmonary congestion with normal blood pressure and transient neutropenia for 96 hours post transfusion.

The clinician should be better aware of TRALI though rare following the blood transfusion. There are numerous risk factors of acute lung injury independent of transfusion (Direct lung injury: e.g. – aspiration pneumonia or Indirect lung injury: e.g., Sepsis, shock).[7] However, TRALI should also be on the differential of acute lung injury when the patient presents with features suggestive of acute lung injury especially following blood transfusion. Early evaluation of preventive actions and in time appropriate target to reduce the risk of TRALI is necessary to prevent its fatal outcome.

The diagnosis of TRALI can be made on the basis clinical suspicion and can prompt to undergo further investigations (CBC, KFT, LFT, Chest Xray, ABG etc.) to support the diagnosis of TRALI.[8] The diagnosis is often by exclusion of other causes of pulmonary edema or ARDS.

Clinical management consists mainly of supportive treatment with diuretics and oxygen therapy via noninvasive ventilation or mechanical ventilation. Mechanical ventilation if needed can be provided in low pressure and tidal volume not to induce barotrauma.[9] We managed our case in non-invasive ventilation. Most of the patients return to their baseline status in few days after acute episode and with timely management as seen in our case.

CONCLUSION

TRALI is a rare life-threatening complication of blood transfusion that is diagnosed clinically. High index of suspicion in diagnosing the case followed by prompt management can prevent fatalities.

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