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Case Report

Transfusion related acute lung injury: a case report

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ABSTRACT

Transfusion Related Acute Lung Injury (TRALI) is one of the most serious complications of blood transfusion. All blood components have been implicated and most often those that contain plasma. The diagnosis is based fundamentally on the integration of clinical, radiological and gasometry elements, once the rest of the possible causes of acute lung injury have been ruled out. The differential diagnosis of a patient who develops a sudden pattern of respiratory failure after a transfusion of blood products must include hemodynamic overload, anaphylactic reaction, bacterial contamination of transfused blood products, haemolytic transfusion reaction and TRALI. Author presented the clinical case of a 33-year-old female patient with grade III hypovolemic shock due to a ruptured ectopic pregnancy, reanimated with crystalloid solutions, globular packages and fresh frozen plasma. The patient developed TRALI for what was managed with ventilatory and hemodynamic support in ICU.

Keywords: Acute lung injury, Anaesthesiology, Blood transfusion, Transfusion

INTRODUCTION

TRALI is defined as an acute lung injury occurring during or within 6h after a transfusion, with a clear temporal relationship to transfusion that is not attributable to another acute lung injury risk factor.[1-3] The frequency of this disease is about 1:5000 patients receiving blood or components, being the fourth most common adverse transfusion reaction and the third leading cause of death associated with transfusion.[4,5] TRAVI has a high mortality rate both in adult and pediatric population (63.7%).[1] The etiology possibly can be caused in the 65-85% of all reported TRALI cases by anti-HLA (human leukocyte antigen) antibodies class I, II, which can be present in the serum of the recipient or donor and react with the donor's or recipient's leukocytes, respectively and by non-immune-mediated TRALI can be attributed to transfusion of biologically active compounds, which are accumulated in stored blood components such as bioactive lipids, proinflammatory cytokines or platelet microparticles with high procoagulant activity.[2]

There are two theories that explain its pathophysiology. The first one called the ''first hit'' is an underlying clinical condition that alerts and prepares the lung

neutrophil, the presence of critical illness and the second one called the ‘‘second hit’’ is transfusion of cellular blood products, that causes the activation of neutrophils in the lung compartment, resulting in TRALI.[1,5,6] There is a common pathway in the pathophysiology of TRALI, the damage to the alveolar-capillary membrane where the neutrophil has been postulated as the protagonist cell for all reactions.[2,7]

Approximately 80% of TRALI cases have been related to the presence of antibodies to human leucocyte antigens or human neutrophil antigens, predominantly in female donor blood.[8] The antecedents of patients with TRAVI include patients with hematologic malignancy undergoing induction chemotherapy major surgery, alcohol dependence, kidney failure, and severe liver disease massive transfusion (replacement of total volume for 4days) and cytokine administration (Granulocyte Colony Stimulating Factor (GCSF), it’s important to know that many clinical studies have shown that patients with more severe underlying disease are at higher risk of developing lung injury after transfusion.[4,8,9]

The clinical presentation is characterized by dyspnea, hypoxemia, arterial hypotension, non-cardiogenic, acute pulmonary edema and fever.[5] During the physical examination the patient showed diffused pulmonary crackles and decreased breath sounds in complementary studies such as chest radiography the patient shows faint, diffuse alveolar infiltrates consistent with pulmonary edema, it’s important not to be confused with other conditions including transfusion-associated circulatory overload (TACO), pneumonia, and acute respiratory distress syndrome (ARDS).[5,10]

CASE REPORT

A 33-year-old woman, G4, P3, who started a day before her admission to the emergency department with colicky, intense abdominal pain located in the right iliac fossa. Go to the service by syncope. Physical examination reveals generalized skin and mucous pallor, tachycardia (125bpm), arterial hypotension (80/60mm Hg) and signs of severe dehydration, as well as changes in alertness, which was why hemorrhagic shock is classified as grade III, secondary to a probable broken ectopic pregnancy. The vaginal cervix is posterior, closed and with little transvaginal bleeding. Resuscitation is started with crystalloid solutions, bladder catheter is inserted without urinary output during the first hours. Laboratory studies are taken with the following results: glucose 493, urea 34, creatinine: 1.9, uric acid 5, albumin: 3, TGO: 16, TGP: 15, hematical biometry: leukocytes 18.3, Hb 4.9, HTO 18, platelets 403 TP: 5.2 and pregnancy immunological test (+). GAS: pH: 6.93, pCO_2: 26, pO_2: 84, HTO: <15%, HCO_3 5.5, BE ef: -26.9. A pelvic USG is performed that reports free fluid in the cavity. It is decided to activate mater code. Author tried to correct the metabolic acidosis based on crystalloid solutions, 89mEq $NaHCO_3$ and 4 globular and 2 plasma packets are transfused. Enter the operating room for exploratory laparotomy. A broken ectopic pregnancy is confirmed, it is decided to practice left salpingectomy+right fibrectomy, 1700ml hemoperitoneum is drained. During the trans-operative period, the patient presented data of respiratory distress and bilateral crepitant rales, wheezing and third noise were not heard. It was decided to take new arterial blood gases and an AP chest X-ray. GAS: pH 7.47, pCO_2 24, pO_2 48, HCO_3 17.5, $Sat0_2$, 85%. The chest radiograph showed diffuse bilateral cottony pulmonary infiltrates and bilateral pleural effusion at both cost-diaphragmatic angles. The patient is intubated and transferred to the intensive care unit. It continues with ventilatory and hemodynamic support.

DISCUSSION

Acute pulmonary damage caused by transfusion or TRALI is a clinical syndrome characterized by acute respiratory failure and non-cardiogenic pulmonary edema during or after a transfusion of blood products.[11] TRALI is defined as acute non-cardiogenic pulmonary edema related to the transfusion of blood products. Non-invasive respiratory support with continuous positive airway pressure (CPAP) or positive airway pressure (BiPAP) may be enough in less severe cases but endotracheal intubation with invasive mechanical ventilation is often required.[12] The clinical case mentions a patient of the fourth decade of life who went to the emergency room due to syncope secondary to hypovolemic shock due to a ruptured ectopic pregnancy. On admission, the patient showed data of grade III hemorrhagic shock, as well as clinical presentation of hemodynamic instability. Prior to the transfusion, the patient did not show respiratory distress, however, there was no chest X-ray prior to transfusion, and arterial blood gas analysis showed metabolic acidosis, apparently secondary to hyperglycemia and shock. Approximately, in a period of no more than 3hours, during the trans-operative period, the patient begins with data of respiratory difficulty, crackles without heart failure. Based on the clinical findings and the background, it is suspected first in pulmonary edema secondary to volume overload. Due to the absence of clinical signs that would indicate that the origin of the edema is cardiogenic, it was decided to treat the condition as a probable TRALI. Supportive therapy is offered based on the ventilatory and hemodynamic requirements. The patient evolves favorably. The blood bank service is notified of the potential risk of the donor transferring antineutrophil antibodies to other patients.

CONCLUSION

TRALI is a complication with high mortality and is poorly diagnosed. Physicians who are in contact with patients who have risk factors to develop TRALI, should be familiar with the clinical manifestations that characterize this entity, in order to act early. This syndrome presents a rapid onset of symptoms, as well as a progressive deterioration of respiratory function.

Despite maintaining adequate supportive treatment, mortality continues to be a problem in both the adult and pediatric population.

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