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MANAGEMENT OF A TRANSFUSION RELATED ACUTE LUNG INJURY (TRALI) IN SPLENECTOMY

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ABSTRACT
TRALI is a life threatening adverse effect of transfusion. It is characterized by dyspnoea , cough and pulmonary edema within 6 hrs of transfusion. Most commonly it is caused by donor HLA antibodies that reacts with recipient antigens . It may also be caused by biologically active compounds accumulated during storage of blood products which are capable of priming neutrophils . Without a gold standard, the diagnosis of TRALI relies on high index of suspicion and on excluding other types of transfusion reactions . We report a case of TRALI in a patient who was posted for splenectomy and required blood transfusion . Patient developed acute pulmonary edema within 1 hour of commencing transfusion . This was managed with post op ventilatory support , Oxygen , Diuretics and Steroid . Patient improved rapidly and later got discharged without any complication.

INTRODUCTION

Transfusion related acute lung injury (TRALI) is a serious blood transfusion complication characterized by the acute onset of non-cardiogenic pulmonary edema following transfusion of blood products. All plasma-containing blood products have been implicated including rare reports of IVIG and cryoprecipitate. It is a rare complication of allogeneic blood transfusion and the incidence has not been well established due to difficulty in defining the syndrome and to variable reporting mechanisms worldwide.

TRALI is associated with a high morbidity with the majority of patients requiring ventilatory support. However, the lung injury is generally transient with PO2 levels returning to pretransfusion levels within 48 -96 hours and CXR returning to normal within 96 hours. TRALI is associated with a significant mortality rate, often approximated at 5 to 10%. Given the gains in safety made within the blood component production industry, particularly with respect to transmission of infectious diseases, TRALI is now among the three leading causes of transfusion related fatalities along with ABO incompatibility and bacterial contamination.

CASE REPORT

A 22 years old male patient presented with complaints of upper abdominal fullness since last 15 days. He had 3 episodes of jaundice in last one month. He was diagnosed to have portal hypertension with splenomegaly and was put on Tablet Propranolol 40mg bd. Examination revealed yellowish discoloration of sclera. Left abdominal lump can be palpated upto 10 cm below costal margin.

Ct scan of upper abdomen revealed – Multiple collaterals in peripancreatic region, splenic vein was dilated and tortuous with diameter of about 20mm. Spleen appears sufficiently enlarged measuring 21cmx18 cmx 12cm in size

As patient's platelet count was 45000/cumm , patient was given 4 Platelet rich concentrates before the day of surgery. Patient was posted for splenectomy with proximal splenorenal shunt surgery. His preoperative vitals were normal . Preoperative investigation Hb-8.14gm%, PC-65000/cumm, INR-1.45, S.bilirubin-2.59mg/dl.

General anaesthesia was planned. Patient was premedicated with Inj Glycopyrrolate 4microgram /kg i/v, Inj Ondansetron 0.15mg/kg i/v, Inj fentanyl 1 microgram/kg i/v. Patient was preoxygenated with 100%

oxygen and induced with Inj Thiopentone 450mg i/v and Inj Succinylcholine 100mg i/v. Patient was intubated with Portex oral cuffed ET tube no 8.5 . Anaesthesia was maintained with Oxygen, Sevoflurane and nitrous. Ventilation was controlled with Inj Vecuronium 0.1mg/kg loading and intermittent doses accordingly.

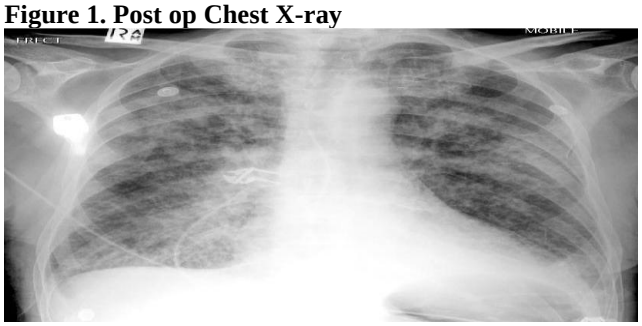
Patient was given 1 litre of Crystalloids and 500ml of Colloid. After splenic artery was ligated , O positive blood was started and two Fresh Frozen Plasma's were given. After all hilum structures were ligated , spleen was removed and surgeons continued with the shunt surgery . Till now surgery was uneventful and all the vital parameters were normal. Blood loss was around 1 -1.5 litres. Then patient's peak airway pressure kept on increasing . It has gone upto >30. Patient's saturation started declining to 93%. ET tube was showing secretions and ET suctioning was done . Initially clear secretions were present in Endotracheal tube . Uptill now surgery was completed and blood loss was around 1 to 2 litres and hemostasis was achieved and there was no ongoing loss but patients blood pressure started falling to 70 mmhg systolic. Second PCV was started at this time. Inj noradrenaline 1 ampoule in 500ml of D5 was started at 20 ml/hr with dial flow. Patients saturation did not improve and now continuous pink frothy secretions were coming out from endotracheal tube. Frequent suctioning was done. Bilateral crepitations were present in upper zone of lungs. Inj Lasix 40 mg was given intravenously and repeated 3 times; total of 120mg of Inj Lasix was given. PCV was stopped. Patient was nebulised continuously with salbuatamol respules.

Decision was made not to extubate the patient and to shift the patient to ICU for mechanical ventilator support.

DAY-1 in ICU- Patient was shifted to ICU and kept on SIMV mode TV-450, FiO2-60%, RR-16, PEEP-5, PSV-10.All necessary investigations were repeated. His Blood Gas analysis was sent at the time of shifting which showed po2-147, pH-7.2, pCO2-61, HCO3-26. Urine output was1000ml Inj noradrenaline was continued at 20ml/hr and blood pressure was maintained above 100mmhg systolic. Inj Lasix and higher antibiotics were started. Inj Midazolam 0.05mg/kg i/v was given intermittently. Post op investigations Hb-9.6gm %, platelet count -1.8 lac. post op Chest X-ray was done which showed infiltrates in bilateral lung fields. A differential diagnosis of fat embolism, Pulmonary edema ARDS was suspected. Frequent suctioning and Nebulisation was done for the first 6- 8 hrs.

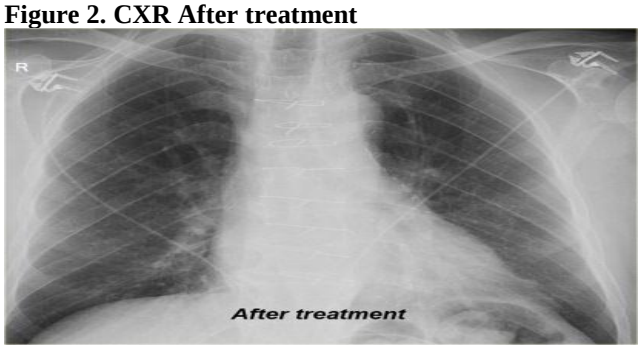
ABG was repeated after 4 hours which showed improvement. Acidosis was resolved and therewas Decline in pCO2 . As Patient became conscious he was shifted to PSV/CPAP mode of ventilation. After 10 to 12 hrs secretions in ETT decreased and crepitations reduced. Blood gas analysis showed marked improvement.

Figure 1. Post op Chest X-ray



DAY 2 IN ICU:- Patient was shifted on T-piece at 4-6litres/min. Abg showed improvement. RS was clear, secretions were reduced, Inj noradrenaline was now decreased to 10ml/hr with dial flow. patient Ddimer and FDP was raised. 4 Fresh frozen plasma's were transfused to correct it. Repeat CBC and CXR showed improvement

Figure 2. CXR After treatment



DAY 3 IN ICU:- Patient was continued on Tpiece ventilation. ABG was normal. Nebulisation and ET tube suctioning was continued

DAY 4 IN ICU:- Patient was weaned and extubated. Inj noradrenaline was stopped. Patient was observed for 1 week in ICU and he remained stable hemodynamically.

DISCUSSION

Transfusion Related Acute Lung Injury is a complex clinical syndrome; it probably does not represent a simple pathogenic entity. There is increasing evidence that this reaction can be triggered by two distinct mechanisms. The subtypes of TRALI based on underlying mechanism into immune (antibody mediated, antiHLA/anti-HNA) or nonimmune (non-antibody mediated, biologically active lipids in stored blood component).

Pathophysiology

The hallmark of acute lung injury (ALI) is that of increased pulmonary microvascular permeability with increased protein in the edema fluid. This is true regardless of the cause of the ALI.

It is hypothesized that TRALI may be precipitated by the infusion of donor antibodies directed against recipient leukocytes. The infusion of donor anti-HLA (human leukocyte antigens) or anti-HNA (human neutrophil antigens) antibodies is thought to directly cause complement activation, resulting in the influx of neutrophils into the lung, followed by neutrophil activation and release of cytotoxic agents, with subsequent endothelial damage and capillary leak.

An alternate hypothesis argues that TRALI is the result of at least two independent clinical events: the first is related to the clinical condition of the patient (infection, cytokine administration, recent surgery, or massive transfusion) that causes activation of the pulmonary endothelium. This then leads to the sequestration of primed neutrophils to the activated pulmonary endothelium. The second event is the infusion of donor derived anti-HLA or anti-HNA antibodies directed against antigens on the neutrophil surface and/or biological response modifiers (e.g., lipids) in the stored blood component that activate these adherent, functionally hyperactive neutrophils, causing neutrophil-mediated endothelial damage and capillary leak.

Yet a third hypothesis suggests that high levels of donor derived vascular endothelial growth factor (VEGF) or antibodies to class II HLA antigens residing on pulmonary vascular endothelium may directly cause endothelial shape change and fenestration. This theory purports to explain the syndrome in neutropenic patients.

CLINICAL FEATURES

Symptoms of TRALI typically develop during, or within 6 hours of a transfusion. Patients present with the rapid onset of dyspnea and tachypnea. There may be associated fever, cyanosis, and hypotension. Clinical examination reveals respiratory distress and pulmonary crackles may be present with no signs of congestive heart failure or volume overload. CXR shows evidence of bilateral pulmonary edema unassociated with heart failure (non-cardiogenic pulmonary edema), with bilateral patchy infiltrates, which may rapidly progress to complete "white out" indistinguishable from Acute Respiratory Distress Syndrome (ARDS).

TREATMENT

Treatment of TRALI is supportive. Mild forms of TRALI may respond to supplemental oxygen therapy. Severe forms may require mechanical ventilation and ICU support. As with ARDS there is no role for diuretics or corticosteroids. The majority of patients recover within 72 to 96 hours and subsequently recover to their baseline pulmonary function without apparent sequelae. However, some patients are slower to recover and may remain hypoxic with persistent pulmonary infiltrates up to seven days. As stated above, approximately 5 to 10% of cases are fatal in spite of aggressive supportive care.

CONCLUSION

In conclusion, correct diagnosis and treatment are very important in TRALI. The keys to the diagnosis of TRALI are high clinical suspicion, differentiation the other possibilities of ALI or Acute Respiratory Distress Syndrome (ARDS), radiological, biochemical and immunological findings. Also TRALI can be developed because of antibodies found in the donor's blood product, it is necessary to know that it may develop as a result of antibodies in the recipient reacting with the donor's leukocytes in the reverse mechanism.

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