Case Report

Transfusion related Acute Lung Injury

Syed Iftikhar Ali[1], Raju C. Ibraham[2], Litty Joseph[3]
Departments of Medicine[1], Anaesthesiology[2] and Gynaecology and Obstetrics[3], Jeddah National Hospital, Jeddah, Saudi Arabia.

Abstract

TRALI (Transfusion related acute lung injury) is a devastating complication of Blood transfusions or its components irrespective of the blood grouping and cross match, We report a case of a Philipino house wife who had blood transfusion for her vaginal lacerations and developed acute pulmonary edema immediately afterwards. Antigranulocyte antibodies were not found in the donors. The patient recovered after supportive treatment. TRALI is not an uncommon complication of Blood transfusion that is under-reported and under-diagnosed.

Introduction

Acute pulmonary oedema has been reported following whole blood transfusions or its components in the medical literature.[1-3] TRALI (Transfusion Related Acute Lung Injury) is a life threatening complication of Blood and its components resembling ARDS (Acute Respiratory distress syndrome) or ALI (Acute Lung Injury). It was first reported in 1992.[4] TRALI is characterized by dyspnoea, hypoxemia, hypotension, bilateral pulmonary oedema and fever despite proper cross matching of blood.[2,4-6] TRALI has been linked to the presence of granulocyte antibodies, HLA class I and II antibodies and biologically active lipids in donor plasma.[6-8] TRALI has been estimated to occur in about one in 5000 transfusions, It usually occurs within six hours of transfusion of Blood or its components or IVIg.[8-12]

Case Report

A twenty-nine years old Filipino housewife (2nd gravida) had a normal vaginal delivery with Right-mediolateral episiotomy. She had postpartum haemorrhage due to vaginal lacerations and was observed in the labour room. Since the bleeding continued, she was taken to the operating room for examination under anaesthesia and sedated with midazolam and ketamine analgesia. On examination the uterus was found to be empty. The bleeding was seen from the raw area of cervix and sutured laceration site after episiotomy. She was transfused with one litre each of polygeline and Ringer's lactate solutions before being given properly cross matched whole blood. The patient complained of severe difficulty in breathing and chest pain and was shifted to the intensive care unit. She was found to have bilateral basal crackles no rhonchi and the JVP was normal. Her BP fell to 80/50, she was conscious and well oriented and her SpO_2 was 82% on pulse oximetry. Blood transfusion was stopped immediately and Oxygen was administered by face mask but the oxygen saturation declined from 82% to 75% during the next few minutes. While the patient was being given oxygen at a rate of 4 litre per minute, a specimen of arterial blood was obtained for evaluating gases. The partial pressure of O_2 was 73 mmHg, the pCO_2 was 42 mmHg and the pH was 7.18. The trachea was intubated and the patient put on mechanical ventilation with 100% oxygen. During this process, her pO_2 was 113 mmHg, pCO_2 36 mmHg and pH 7.27. The 12 lead ECG showed only sinus tachycardia and no ST segment or T wave changes. A portable radiograph of the chest revealed diffuse bilateral interstitial shadowing consistent with pulmonary oedema. At this time the patient has stopped bleeding from the cervix.

Urgent evaluation of the blood revealed no evidence of reaction to the transfusion. The temperature rose to 37.9^0C and subsided after six hours. An urgent echocardiogram revealed no abnormalities of the heart valves neither hypokinesia nor dyskinesia of the myocardium. The patient was given iv methylprednisolone, iv Dopamine infusion, iv ceftriaxone and iv mannitol. She was on assisted ventilation for twenty-four hours and made an uneventful recovery. The medications were stopped on 5th day without any untoward incident. Her lab reports were within normal limits except a prolonged APTT and haematuria. Blood culture for aerobic and anaerobic organisms was negative after 3 days. The donors for the patient were male phillipino subjects whose sera were devoid of any antigranulocyte antibodies at a look back

investigation. Her levels of factor VIII and IX were within normal limits. We were unable to ascertain the cause of bleeding in this patient with prolonged APTT. Whether it was due to dilutional coagulopathy is a question unanswered.

Discussion

Cases of TRALI have been observed in the past but the entity has very recently been recognized and reported.[6-10]

Table of Investigations.

Variable	Unit	Prior to admission	Day on admission	Day II	Day III
Hb	g/dl	12.4	10.1	10.6	10.4
Hct	%		30	33	31
MCV	Fl		81	77	78
MCH	Pg		26	25	26
MCHC	g/dl		32	32	32
TLC	10*9/L		15.6	16.4	15
Neutr	%		89	90	88
Lympho	%		9	5	7
Eosinop	%		1	1	1
Platelets	10*9/L		212	154	150
ESR	mm/1sth		26	28	28
Glucose R	mg/dl		104	150	170
Urinalysis					
Glucose		nil	nil	nil	nil
Protein		nil	nil	nil	nil
Bilirubin		nil	nil	nil	nil
Blood		nil	present	present	nil
WBC		nil	2-4	2-4	1-3
RBC		1-2	6-8	1-5	1-3
Blood Group			O positive		
HCV		Negative			
HBsAg		Negative			
HIV		Negative			
APTT	sec	56	60	38	32
PT	sec	17	17	17	17
BT	min	3			
Urea	mg/dl	20	36	40	
Creatnine	mg/dl	1.1	1.43	1.2	
ANA	EU/ml	Negative			
T.Bilirubin	mg/dl		0.8		
SGPT	u/L		20		
SGOT	u/L		17		
LDH	u/L		150		

Usually the donors implicated in TRALI cases are multiparous females[5] but it has also been reported after blood transfusion by male donors.[4] In the presented case it was male donors with the same ethnic background and no anti-granulocyte antibodies in the sera. Although the pathogenesis of TRALI is unknown, there is sufficient evidence to implicate an immune reaction and unlike most immunologially mediated immune reactions the pathologic antibodies in TRALI originate from donors rather than the recipients. Antibodies in the donor suspected to be the causative agent because the 'substrate' is the recipients extracirulatory system and marginated pool of leucocytes. Since there is no diagnostic test nor any pathognomonic sign of TRALI it is a diagnosis of exclusion.

Other causes of respiratory distress and pulmonary oedema in patients receiving blood transfusions should be excluded. Myocardial infarction, Circulatory overload and bacterial infections or any other causes of ARDS notably SARS should be considered. Normal Jugular venous pressure is consistent with the diagnosis of TRALI. The treatment of TRALI is symptomatic.[5] Presor agents may be useful for sustained hypotension, corticosteroids are of marginal value and diuretics have no role because it is a micro vascular injury rather than fluid overload.[13] However, in this case mannitol and steroids were used. Chromosomal analysis was not done in our case. The question why the lung is the primary end organ of choice is not answered.[5] It is recommended that if a TRALI case occurs, the donor should be temporarily suspended from donation for three months and 14 ml of clotted and 7 ml EDTA blood obtained from the donor (fresh samples of whole blood should be obtained as plasma is not appropriate) and sent for HLA type I and II antibodies.[4,5] TRALI incidence is much higher than is thought of and it is still an under diagnosed and under reported illness.[11-13]

References

1. Levy GJ, Shabot MM, Hart ME et al.: Transfusion-associated noncardiogenic pulmonary edema. Transfusion 1986;26:278-81.
2. Chung YT, Wu YC, Chen YH : Postoperative pulmonary edema, transfusion-related?--a case report Acta Anaesthesiol Sin. 2003;41:43
3. Popovsky MA, Chaplin HC, Moore SB. Transfusion-related acute lung injury: a neglected, serious complication of hemotherapy. Transfusion 1992;32:589-592.
4. Smith N,Copplestone A,Gesinde M,MacLennan S, Morgan C MacLennan, Mortimer AJ et al:Transfusion related acute lung injury(TRALI):a monograph approved by the National Blood Service Transfusion Medicine Clinical Policies Group on march 5,2003 accessed on internet on 5/9/2003.
5. Kopko PM, Marshall CS, MacKenzie MR, Holland PV, Papovsky MA : Transfusion related acute lung injury (report of a clinical look back investigation) J Am Med Assoc 2002;287:1968-71
6. Kopko PM, Holland PV: Transfusion related acute lung injury.Br J Haematol 1999;105:322-8
7. Voss J, Westphal K, Böhme J, Bux J, Greinacher A : The TRALI syndrome-a life-threatening transfusion reaction: Anaesthesist 2001 50:930-2.
8. Silliman CC, Boshkov LK, Mehdizadehkashi Z, Elzi DJ, Dickey WO, Podlosky L, et al. Transfusion-related acute lung injury: epidemiology and a prospective analysis of etiologic factors. Blood 2003. 101:454-62.

9. Semple JW, Kim M, Lazarus AH, Freedman J Gamma-globulins prepared from sera of multiparous women bind anti-HLA antibodies and inhibit an established in vivo human alloimmune response: Blood. 2002;100:1055-9.

10. Kopko PM, Popovsky MA, MacKenzie MR, Paglieroni TG, Muto KN, Holland PV: HLA class II antibodies in transfusion-related acute lung injury. Transfusion 2001, 41:1244-8.

11. Win N, Montgomery J, Sage D, Street M, Duncan J, Lucas G. Recurrent transfusion-related acute lung injury :Transfusion 2001,41:1421-25.

12. Wallis JP, Lubenko A, Wells AW, Chapman CE. Single hospital experience of TRALI. Transfusion 2003, 43:1053-9.

13. J. P. Wallis. Transfusion-related acute lung injury (TRALI)-under-diagnosed and under-reported. Br J Anaesth 2003;90:573-6.