

# Management of mismatched blood transfusion – a case report

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## Abstract

Complications of blood transfusion are rare but can be life-threatening. Since 2005, it has been a legal requirement that all serious adverse reactions attributable to the safety or quality of blood are reported. Most reported complications are because of transfusion of mismatched blood products and are avoidable through clinical vigilance. We report the case of a 23 yr old male patient who underwent uneventful orthopedic surgery and received accidental mismatched blood transfusion in the ward. This patient was managed in the critical care unit with forced alkaline diuresis.

## CASE-REPORT

A 23 yrs old male patient was admitted in orthopedic ward with posttraumatic fracture L<sub>1</sub> compression with paraplegia. His preoperative hemoglobin was 11.2gm%. After thorough preoperative workup the patient underwent pedicle screw fixation. Intraoperative course was uneventful with blood loss around 200ml and the patient was extubated and shifted to ward. No blood was issued for the patient.

In the ward a blood transfusion was started. The patient started having fever, chills and headache on receiving 100ml of blood. The transfusion was immediately stopped. On checking the blood bag label it was found that blood had been started to the wrong patient accidentally by staff-nurse without confirming blood receiving/checking notes. The patients' blood group was O RH positive and he had received 100ml AB RH positive blood.

A diagnosis of mismatched blood transfusion been made, patient was given Inj.Hydrocortisone 100 mg IV, Inj.Avil 2cc IV and Inj. Frusemide 20mg IV stat. By then the patient had developed hematuria and complained of headache. There were no complaints of rigors, rash, breathlessness, edema or bleeding from abnormal sites. On examination, pulse rate – 96/min, BP – 160/100 mmHg. The patient was put on ventimask with O<sub>2</sub> @ 4 lit/min and shifted to the critical care unit (CCU) for monitoring and further management.

In the CCU the patient was put on ventimask with O<sub>2</sub> @ 4 lit/min with FiO<sub>2</sub> 60%. In CCU, pulse rate – 50/min, BP –

96/50 mmHg, SpO<sub>2</sub> - 99%. ABG on admission revealed: p<sup>H</sup> – 7.455, pO<sub>2</sub> – 135.5 mmHg, pCO<sub>2</sub> – 37.1 mmHg, HCO<sub>3</sub> – 25.8 mmol/L. Sr.K<sup>+</sup> - 4.24 mmol/L. Inj. Frusemide 10 mg/hr drip was started. Inj.Mannitol 1.5 gm/kg was given IV. Blood samples were withdrawn and sent to the blood bank for grouping and cross matching.

Central venous access was secured in right cephalic vein with 14x16 central venous catheter. CVP was 1-2 cm of H<sub>2</sub>O. Inj. Dopamine drip (800 mg in 500 ml normal saline) was started @ 16 µdrops/min. Forced alkaline diuresis was initiated as per the following protocol given by the nephrologist :

1<sup>st</sup> cycle - 1 pint normal saline + 20mEq sodium bicarbonate over 1 hr followed by I pint normal saline over 1 hr, further followed by Inj. Frusemide 60mg IV. Send urine for Hb and myoglobin.

2<sup>nd</sup> cycle – Repeat same as above over 2 hrs.

3<sup>rd</sup> cycle – Immediately repeat same cycle over 4 hrs

Inj. sodium bicarbonate to be given 60 mEq divided in 3 cycles. After completing 3 cycles repeat Hb, CBC, urine for routine, microscopy and myoglobin, Sr. Urea, Sr. Creatinine, Sr.electrolytes..

The patients' blood samples were sent for all routine investigations. 3 cycles of forced alkaline diuresis were given. CVP was maintained at 7 – 8 cm H<sub>2</sub>O. BP was maintained > 90 mmHg systolic with dopamine drip.

Hematuria gradually decreased. NSAIDs were withheld.

Investigations were as follows:

**Figure 1**

Investigation	Day 1	Day 2	Day 3	Day 4	Day 5
Hemoglobin (gm/dl)	10	11.1	9.7	11.6	
Total leucocyte count (per cu.mm)	12000	11400	18000	9600	
Differential leucocyte count	Neutrophils 90 Leucocytes 8 Monocytes 1 Eosinophils 1	Neutrophils 76 Leucocytes 22 Monocytes 1 Eosinophils 1	Neutrophils 60 Leucocytes 34 Monocytes 2 Eosinophils 4	Neutrophils 68 Leucocytes 30 Monocytes 1 Eosinophils 1	
Platelets	Adequate	Adequate	3,39,000/cu.mm	Adequate	
Prothrombin time	Test 17 control 12	Test 15 control 12	Test 14 control 12	Test 15 control 12	Test 12 control 12
INR	1.0	1.4	1.3		1.1
APTT					Test 30 control 26
Random blood sugar (mg/dl)	106	86	76	108	106
Sr. Urea (mg/dl)	38	59	44	39	31
Sr. Creatinine (mg/dl)	0.9	1.2	0.7	0.7	0.7
Sr. Alkaline Phosphatase (IU/L)	94	87	68		62
Sr. Bilirubin (mg/dl)	0.5 Direct – 0.2 Indirect – 0.3	0.8	0.6		
SGOT (IU/L)	25	57	30		32
SGPT (IU/L)	17	22	18		20
Sr. Proteins (mg/dl)	6.9 Albumin – 3.8 Globulin – 3.1	5.8 Albumin – 3.5 Globulin – 2.2	5.4 Albumin – 3.3 Globulin – 2.1		5.6 Albumin – 3.3 Globulin – 2.3
Sr. Na <sup>+</sup> (mEq/lit)	141		132.2		
Sr. K <sup>+</sup> (mEq/lit)	4.2		3.94	3.92	3.78
Sr. Cl <sup>-</sup> (mEq/lit)	102				
Sr. Ca <sup>++</sup> (mEq/lit)	9.6				
Sr. PO <sub>4</sub> (mEq/lit)	3.4				
LDH (IU/L)		1038			
Direct Coombs test		Weakly positive			
CT scan brain			Essentially normal study		
Fundoscopy			normal		
Arterial blood gas analysis	pO <sub>2</sub> – 135 pCO <sub>2</sub> – 37.1 p <sup>H</sup> – 7.455 HCO <sub>3</sub> – 25.8		pO <sub>2</sub> – 190.8 pCO <sub>2</sub> – 41.1 p <sup>H</sup> – 7.441 HCO <sub>3</sub> – 26.7	pO <sub>2</sub> – 179.5 pCO <sub>2</sub> – 35.5 p <sup>H</sup> – 7.498 HCO <sub>3</sub> – 27.7	pO <sub>2</sub> – 95 pCO <sub>2</sub> – 38.7 p <sup>H</sup> – 7.46

Peripheral smear examination on day 1 revealed hypochromic RBS's. All counts were reduced on PS. Very few normochromic RBC's were seen. On day 5, PS showed microcytes, target cells and anisocytes. Retic count was

1.5%. Direct Coombs test was weakly positive. Serum LDH was greatly elevated. Urine routine and microscopy was normal except for occasional uric acid crystals. CT scan of brain was normal. BP remained on the lower side for 3 days but vitals were stable by day 5. The patient was shifted to ward on day 6 and eventually discharged.

## DISCUSSION

Transfusion reaction accompanies or follows intravenous administration of blood components. Its severity varies from mild (fever and chills) to severe (acute kidney failure or complete vascular collapse and death), depending on the amount of blood transfused, the type of reaction, and the person's general health

Hemolytic reactions (red blood cell rupture) follow transfusion of mismatched blood. Transfusion with incompatible blood triggers the most serious reaction, marked by intravascular clumping of red blood cells. The recipient's antibodies (immunoglobulin G or M) adhere to the donated red blood cells, leading to widespread clumping and destruction of the recipient's red blood cells and, possibly, the development of disseminated intravascular coagulation and other serious effects

Allergic reactions are fairly common but only occasionally serious. Febrile nonhemolytic reactions, the most common type of reaction, apparently develop when antibodies in the recipient's plasma attack antigens.

Immediate effects of hemolytic transfusion reaction develop within a few minutes or hours after the start of transfusion and may include chills, fever, hives, rapid heartbeat, shortness of breath, nausea, vomiting, tightness in the chest, chest and back pain, low blood pressure. bronchospasm, angioedema, and signs and symptoms of anaphylaxis, shock, pulmonary edema, and congestive heart failure. In a person having surgery under anesthesia, these symptoms are masked, but blood oozes from mucous membranes or the incision.

Delayed hemolytic reactions can occur up to several weeks after transfusion, causing fever, an unexpected decrease in serum hemoglobin, and jaundice.

Allergic hemolytic reactions typically don't cause a fever and are characterized by hives and angioedema, possibly progressing to cough, respiratory distress, nausea and vomiting, diarrhea, abdominal cramps, vascular instability, shock, and coma.

The hallmark of febrile nonhemolytic reactions is a mild to severe fever that may begin when the transfusion starts or within 2 hours after its completion.

Confirming a hemolytic transfusion reaction requires proof of blood incompatibility and evidence of hemolysis. When such a reaction is suspected, the person's blood is retyped and cross matched with the donor's blood.

At the first sign of a hemolytic reaction, the transfusion is stopped immediately. Depending on the nature of the person's reaction, the health care team should:

- report “transfused incompatible blood” to the medical staff and ask them to help
- stop transfusion and begin fluid infusion
- monitor vital signs every 15 to 30 minutes, watching for signs of shock
- maintain an open intravenous line with normal saline solution, insert an indwelling urinary catheter, and monitor intake and output
- cover the person with blankets to ease chills
- deliver supplemental oxygen at low flow rates through a nasal cannula or hand-held resuscitation bag (called an AMBU bag)
- check for signs of DIC

Administer drugs such as intravenous medications to raise blood pressure and normal saline solution to combat shock, Adrenaline to treat shortness of breath and wheezing, Benadryl to combat cellular histamine released from mast cells, corticosteroids to reduce inflammation, and Osmitol or Frusemide to maintain urinary function. Parenteral antihistamines and corticosteroids are given for allergic reactions (anaphylaxis, a severe reaction, may require Adrenalin). Drugs to reduce fever are administered for febrile nonhemolytic reactions and appropriate intravenous antibiotics are given for bacterial contamination.

At the same time, the following examinations should be performed:

1) Reexamine blood type of both patient pre-transfused blood sample and donor blood;

2) Check hemolysis, renal function, and DIC.

Transfusion errors are mainly due to transfusion to a wrong patient or transfusion of a wrong blood bag. Therefore, to prevent ABO-incompatible transfusion, identification of the patient and the blood bag are very important before transfusion

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