



RESEARCH ARTICLE

MANAGEMENT OF MISMATCHED BLOOD TRANSFUSION REACTION
BY FORCED ALKALINE DIURESIS

Dr. Dheeraj Singha, Dr. Priyanka Sood, *Dr. Aman Thakur and Dr. Sudarshan Kumar

Department of Anaesthesiology, Dr. Rajendra Prasad Govt. Medical College, Kangra at Tanda

ARTICLE INFO

Article History:

Received 21st February, 2017
Received in revised form
11th March, 2017
Accepted 16th April, 2017
Published online 23rd May, 2017

Key words:

Anaesthesia, Caesarean section,
Blood transfusion reaction,
Forced alkaline diuresis.

ABSTRACT

In developing countries people are more worried about infectious risks of blood transfusion but various reports show that antigen-antibody reactions are responsible for vast majority of transfusion related complications, most common and serious being intravascular haemolytic transfusion reactions because of ABO incompatibility caused by giving wrong blood to the patient. We report a case of 24 year old female patient who underwent emergency caesarean section for foetal bradycardia and was accidentally given mismatched blood transfusion in preoperative period. This patient was managed subsequently after the emergency surgery in the Intensive care unit.

Copyright©2017, Dr. Dheeraj Singha et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Citation: Dr. Dheeraj Singha, Dr. Priyanka Sood, Dr. Aman Thakur and Dr. Sudarshan Kumar, 2017. "Management of mismatched blood transfusion reaction by forced alkaline diuresis", *International Journal of Current Research*, 9, (05), 50465-50467.

INTRODUCTION

Blood transfusion reaction can be defined as any adverse effect or an undesirable and unintended occurrence during or after transfusion of blood component/product. These adverse effect may manifest as fever and hives to more serious complications like renal failure, shock and even death. Blood transfusion reactions can be classified as acute or delayed depending upon time of onset and immune or non immune mediated depending upon pathophysiological mechanism. Transfusion of blood has been found to be a safe and effective way to correct haematological deficits. But at the same time it is also important that blood should be used in a judicious way and health care provider must be aware of risks associated with blood transfusion.

Case report

A 24 year old female (weight 45 kg, height 150 cm) was posted for emergency caesarean section in view of fetal bradycardia with previous history of Caesarian section. During pre-anesthesia checkup it was found that patient was having severe anemia with Haemoglobin of 4.7 gm %. Since it was an emergency surgery, patient was immediately shifted to emergency operation theatre with the ongoing blood transfusion which was started by an intern in the ward.

*Corresponding author: Dr. Aman Thakur,
Department of Anaesthesiology, Dr. Rajendra Prasad Govt. Medical College, Kangra at Tanda.

Patient's pre-operative vitals were HR - 118/min, BP - 98/56 mmHg and oxygen saturation - 98% on room air. As the patient was severely anaemic, General Anaesthesia with Rapid Sequence Induction was planned. In the operation theatre, ECG, noninvasive blood pressure and a pulse oximeter were attached. Induction was done with thiopentone 100mg slow iv, ketamine 40 mg iv and succinylcholine 75 mg iv. Rapid Sequence Induction was done with cricoid pressure and tracheal intubation performed. Maintenance of anesthesia was done using oxygen 33%, nitrous oxide 66%, halothane 0-0.5% Atracurium 20mg and fentanyl 60 microgm (which was administered after delivery). Intraoperatively red coloured urine was noticed and there was generalised ooze from the surgical field. Blood transfusion was immediately stopped and inj. Hydrocortisone 200mg and inj. Chlorpheniramine 10mg iv was administered immediately. Fluids were rushed to maintain blood pressure and injection furosemide 20 mg iv given. Vitals remained stable throughout the intraoperative period. Baby delivered was normal with 8/10 apgar score. Patient was shifted to ICU post-operatively and placed on ventilator with Fio2 60%. Patient vitals were as follow HR 98/min, BP 99/39, and oxygen saturation 99%. Central venous access was secured in right internal jugular vein and CVP was found to be 5 cm of H₂O. Forced diuresis was planned. 3 cycles were planned (one cycle for 2 hours), each cycle consisted of giving 500ml of Normal Saline with 20 mEq of Sodium bicarbonate followed by giving Inj. Furosemide 60 mg iv. After each cycle urine was analysed for Haemoglobin and pH and blood for

Table 1. Postoperative ABG of patient on successive days

	pH	PaO ₂ (mm of Hg)	PaCO ₂ (mm of Hg)	HCO ₃ (mmol/L)	BE (mmol/L)	Spo ₂ (%)	Na (mmol/L)	K (mmol/L)	Ca (mmol/L)
1st post operative day	7.34	140	38	20.5	4.8	99	136	4	3.25
2 nd post operative day	7.38	98	34	21	4.9	97	137	3.5	3.2
3 rd post operative day	7.37	146	32	18.5	3.7	99	139	3.7	3.01
4 th post operative day	7.37	177	34	19.9	4.6	100	139	4	3.65

Table 2. Postoperative investigations of patient on successive days

	Hb(gm%)	RBS(mg)	Platelets	Urea(mg/dl)	Creatinine (mg%)	PTI	INR	Bilirubin(mg/dl)
1st post operative day	4.0	109	47000	25	0.5	50.6	2.17	3.06
2nd post operative day	5.5	143	98000	72	1.0			4.5
3 rd post operative day	6.4	128	124000	166	3.4			2.4
4 th post operative day	8.3	131	129000	128	3.2	81	1.35	2.6

potassium level. During each cycle CVP was maintained around 8-10 cm of H₂O. Mean Blood Pressure was maintained more than 60mm of Hg. If required Dopamine was started at the rate of 5 to 10 microgm /kg/min.

Blood samples were sent for ABO-Rh typing, Direct coombs test and indirect coombs test, urinalysis, coagulation profile, Complete haemogram, Renal Function Tests and serum electrolytes. Blood group was O positive, Direct coombs test was negative and urinalysis revealed proteinuria 3+ and erythrocytes 4 +. Coagulation profile was found to be grossly deranged (PTI 50.6% and INR 2.17). FFP and fresh blood transfusions were done, subsequent transfusions were of O positive blood group (Total 7 units of O positive blood given). Next day, urine was found to be clear. Also, patient was weaned off from mechanical ventilation next day. RFTs were deranged on 1st post operative day and urea creatinine kept on rising till 3rd post-operative day with value of urea being 166 and creatinine 3.4. After that renal functions kept on improving and we were able to shift patient to obstetrics ward on 4th postoperative day. Patient was then discharged from hospital after full recovery.

DISCUSSION

As infectious complications from blood transfusion have decreased because of improved donor questionnaires and sophisticated infectious disease blood screening, noninfectious serious hazards of transfusion (NISHOTs) have emerged as the most common complications of transfusion (Hendrickson and Hillyer, 2009). This case provide us with unique opportunity for the early diagnosis and overall management of acute blood transfusion reaction. High degree of alertness and suspicion should be maintained during blood transfusion and any adverse acute clinical event should be attributed to blood transfusion until proven otherwise. Acute hypotensive transfusion reaction is characterized by an early and abrupt onset of hypotension, which is often severe, other than signs or symptoms that are attributed directly to the drop in systolic blood pressure. Once the transfusion is stopped, the hypotension rapidly resolves without specific therapy (Popovsky, 2001). Presence of Hypotension may be the first principal event to occur during mismatched blood transfusion in an intubated patients. It is a common and non-specific feature of acute haemolysis, severe allergic reaction, bacterial contamination or TRALI (Kopko and Holland, 1999). It occurs rarely as an isolated finding and some cases have been attributed to the generation of bradykinin and angiotensin when blood components were exposed to charged surface of leucoreduction filters. It may

also be present with other clinical reaction as found in a French study where 3 cases out of 4 transfusion induced hypotension were associated with other clinical reaction. Beside hypotension in an intubated patient, blood transfusion reaction may present with unexplained tachycardia and increase in airway pressure.

In our case blood transfusion reaction was first suspected by presence of red colored urine in the intra operative period. Kamitani and Sakai had already reported, recognition of blood transfusion reaction by occurrence of haematuria during intra operative period. There was generalized ooze at the operative site which was noticed by the surgeon, similar findings were also reported in another study. Off late the mechanism for such a diffuse bleeding is due to glycoprotein A antibodies which may induce lipid bilayer exposure and cation permeability independent of agglutination as suggested by Garratty. Immediately after confirmation of haemolytic transfusion reaction the transfusion should be stopped while keeping intravenous access available, start intra venous fluids preferably Normal Saline. Consider placement of Central line. Check the label and report it as "incompatible blood" to the medical staff. Insert indwelling urinary catheter for input output monitoring. A new blood sample from the patient (free of hemolysis), should be sent along with the remainder of the infused product to the blood bank for inspection and analysis. Monitor for the sign of DIC and shock. Parenteral antihistamines and corticosteroids can be administered for allergic reaction and Adrenaline may be required for severe anaphylactic reaction. Furosemide may be administered to increase Renal flow which should be maintained at 30-100ml/hr. Forced alkaline diuresis which results in increase urine formation can be started in the ICU. It is important to monitor potassium levels during this procedure. Renal insufficiency and cardiopulmonary decompensation are contraindications for this procedure.

REFERANCES

- Garratty G. 2010. A new mechanism for immune destruction of red blood cells? *Transfusion*. 50: 274-277.
- Hendrickson JE. and Hillyer CD. 2009. Noninfectious serious hazards of transfusion. *Anesth Analg.*, 108(3):759-69.
- Kamitani T. and Sakai T. 2007. Reaction to blood transfusion recognized by sudden onset of red urine during operation. *Masui*. 56(7):847-9.
- Klein H, Spahn D, Carson J. 2007. Red blood cell transfusion in clinical practice: *The Lancet*; 370: 415-426.
- Kopko P. and Holland P. 1999. Transfusion-related lung injury. *BR J Haematol.*, 105: 322-329.

- Popovsky MA. 2001. Transfusion Reactions. Bethesda, MD: AABB Press; 222.
- Py JY, Leo-Kodeli S, Fauveau L, Duedari N, Roubinet F. 2009. Hypotension and adverse transfusion reactions: from the associated clinical signs to the hypotensive transfusion reaction. *Transfus Clin Biol.*, 16(1):12–20.
- Reddy M, Sriganesh K, Bhadrinarayan V, Raghavendra BS. 2011. Unusual Manifestation of Blood Transfusion Reaction as Diffuse Operative Site Oozing, Hypotension and Brain Swelling. *J Anaesthesiol Clin Pharmacol.*, 27(1): 130–31.
