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Case Report

Successful Management of ABO Mismatched Blood Transfusion: A Challenging Clinical Scenario- A Case Report

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ABSTRACT

The complications of blood transfusion are known and at times is life threatening especially when it occurs due to accidental major mismatched blood transfusion. We report a case of anemic lady delivered by elective LSCS for scar tenderness who received erroneous transfusion of group A Rh-negative blood while her actual group is O Rh-negative during immediate post LSCS period. We rushed her to critical care unit and saved her life. We hope that this presentation will help those who may have to face similar grave situation.

Keywords: Blood group Incompatibility, Blood transfusion, Critical care, Forced alkaline diuresis.

INTRODUCTION

The ABO and Rh blood group system is the most important system which is been followed prior to blood transfusion. [1] Also rhesus blood group system is the second most important blood group followed during blood transfusion. [2] Blood group O is the commonest which is 37.12%. Further, it is followed by B which is 32.26%, followed by A which is 22.88% and AB which is the least prevalent group at 7.74%. Similarly out of the total donor population, 94.61% are Rh (D) positive and rest 5.39%, are Rh (D) negative. [3] Most recipients of ABO incompatible blood have blood group O [4] and as in our case too. Acute transfusion reactions (ATR) are either immunological reactions or nonimmunological reactions or both. Out of which only the hemolytic reaction of immunological type was found and managed successfully in this case.

CASE REPORT

A 28 year old anemic lady of second gravida with scar tenderness of previous LSCS was admitted on 14th July 2014. Her routine workup showed Hb-9.4 gm%, TLC-6180c/cmm, DC- N-90%, L-10%, E-0%, BT-2'.00", CT-5'.30'', Blood group- O Rh negative. As per standard protocol before LSCS, one pint of matching blood was arranged on 15th July 2014. She underwent LSCS under regional anesthesia uneventfully on 15th July 2014 at 4.15 PM. Intra operatively there was minimal blood

loss of approximately 450 ml, clear urine output approximately of 250 ml, and all vital signs well within normal limits. Surgery was finished at 4.45 PM and patient was shifted to ward at 5.00 PM. Patient remained conscious and well oriented while back in ward. In view of preoperative anemic condition, intra operative blood loss, and for better wound healing postoperatively one pint of already cross matched blood was called in from the blood bank and transfusion started at 7.00 PM. The blood transfusion was finished at11.45 PM but by then she had nausea, 3 bouts of hematemesis, frank hematuria and blood soaked abdominal wound dressing. She remained fully conscious and well oriented with no breathlessness, cyanosis or sweating. Vital signs at this stage showed as pulse-80/min., BP- 130/90 mm of Hg. She had dark red colored urine of about 500 ml, and minimal per vaginal bleeding. The uterus was well contracted and retracted.

The possibility of blood transfusion reaction was suspected, and therefore, on immediate further enquiry it was found that the group A Rh- negative blood was transfused erroneously instead of O Rh-negative group of the patient. It was also found that there occurred serious lapses in checking the blood bag by the duty medical officer as well as by the staff nurse before actual starting of the transfusion due to over site- a human error, an obvious negligence.

Immediately the patient was rushed to critical care unit for further management at 1.45 AM on 16th July 2014 (Day 0). Patient remained conscious, oriented, afebrile and with stable vitals on arrival to critical care unit i.e. her pulse was 78/ minute, BP- 100/60 mm of Hg. In view of history of erroneous one complete pint of mismatched blood transfusion and subsequent active hematuria, active fresh oozing of blood from operative site and recent history of hematemesis the provisional diagnosis of major mismatched blood transfusion reaction confirmed and immediate treatment with forced alkaline diuresis started, the details are as following:

On day 0:

1. Nil by Mouth
2. 0.9 % Normal saline totals of 8 pints each containing 500 ml
3. One ampoule containing 7.5% (44.6 mEq.) of Sodium bicarbonate in alternate pint of normal saline
4. 10 mg of Furosemide in alternation with sodium bicarbonate in pints of normal saline

In addition to above, patient received following supportive treatment on day-1, 2, 3 and 4:

1. 100 mg of Hydrocortisone hemisuccinate IV every 8th hourly
2. Injection Ceftriaxone 1 Gm IV every 12th hourly
3. Injection Metrogyl 500 mg IV every 8th hourly
4. Oxygen by ventimask @ 2 liters/minute

A complete routine investigation work up was made on arrival to critical care unit (refer day-0 table- 1, 2a and 3). Specific investigations such as tests for liver function, renal function, hemoglobunuria, INR and Coombs test were carried out (refer day-0 table- 4).

- Blood sent for grouping and cross matching
- Urine pH was maintained at 8
- Obstetric examination revealed well contracted and retracted uterus, blood soaked abdominal wound dressing
- Total IV input-4000 ml. and dark red urine output-600 ml

Tables of investigation details:

Table-1: Hemogram

Work up	Day-0	Day-1	Day-3	Day-6	Day-10	Day-11
Hb %	9.4	7.7	7.1	6.5	8.7	10.5
TLC	6180	20690	17700	14760	12600	14520
Neutrophils Lymphocytes Eosinophils	90% 10% 0	89% 11% 0	89% 11% 0	88% 10% 02%	80 20	82 15 03
Platelets	1.20	1.20	1.95	2.10	2.10	2.55
MCV	89.1	90.2	91.6		42%	
PCV	29.4%	23.0%	21.7%	19.9%	26.8%	
BT	2'00"					
CT	5'30"					

Table-2a: Biochemical tests

Work up	Day-0	Day-1	Day-2	Day-3	Day-4	Day-5	Day-6	Day-7
BSL mg/dl	83			96	61			91
Ser. Creat. mg/dl	0.9	2.1	2.8	3.5	3.7	4.2	3.9	2.6
BUN mg/dl	29	85	116	130	124	121	109	63
Na^{++} mEq/l	138		139	138	139			139
K^{++} mEq/l	4.0		3.6	3.6	3.6			3.0
Pro-time- Patient Control	 19 13	 13 13	 13 13			 17 13	 16 13	
INR	1.7	1.0	1.0			1.4	1.3	

Table-3: Urine analysis

Work up	Day-0	Day-1	Day-2	Day-9
Urine Routine				
Color	Red	Light Red	Pale Yellow	Pale Yellow
Sp Gravity	1.015	1.015	1.015	1.005
Appearance	Turbid	Turbid	Turbid	Clear
pH	8.0	7.9	8.0	6.0
Albumin	++	++	++	+
Sugar	Nil	Nil	Nil	Nil
Urine Microscopy				
WBC	8-10/HPF	7-8/HPF	8-10/HPF	4-5/HPF
RBC	0-1/HPF	0-1/HPF	0-1/HPF	Nil
Hemoglobinuria	Present	Present	Absent	Absent
Epithelial Cells	1-2/HPF	3-4/HPF	5-6/HPF	5-6/HPF

On day 1:

The general condition and the obstetric examination showed status quo. She had pallor, icterus, non tender soft abdomen, normal peristalsis. She was encouraged to take oral fluids freely.

Second cycle of forced alkaline diuresis continued similar to on day-0.

In addition to above supportive drugs patient received -Hematinics, Folvite.

On 17/07/2014 investigation work up revealed (refer day-1 in table- 1, 2a and 3):

- Drop in levels of hemoglobin and prothrombin-time
- Raised serum creatinine and blood urea nitrogen
- Urine pH 8
- Total IV+ oral input-5000 ml. and light red urine output-1600 ml.

On day 2:

The general condition and obstetric examination showed status quo. The abdomen was soft, non tender, normo peristaltic. She tolerated; oral fluids and was further started with soft oral diet.

Third cycle of forced diuresis continued as following:

1. 0.9 % Normal saline totals of 4 pints at rate of 500 ml every 6 hourly
2. 1 Ampoule of Sodium bicarbonate in alternate pint of normal saline
3. 20 mg of Furosemide in alternation with sodium bicarbonate in pints of normal saline.

The supportive treatment mentioned above was continued on day-2 and 3.

On 18/07/2014 investigation work up revealed (refer day-2 in table-1, 2a and 3):

- Un changed values of serum electrolytes and prothrombin-time
- Raised serum creatinine and blood urea nitrogen
- Urine pH 8
- Total IV + oral input-3000 ml. and clear urine output-2200 ml.

In view of rising serum creatinine and urea and drop in hemoglobin level due to hemolysis, the hemodialysis and matching blood transfusion was planed.

On day 3:

The general condition and obstetric examination revealed status quo. Pallor and icterus was persistent. She was allowed to take full oral diet freely.

Fourth cycle of forced diuresis continued as following:

1. 0.9 % Normal saline totals of 7 pints
2. 20 mg of Furosemide in every alternate pints of normal saline

In addition to above supportive treatment on day-3 and 4 patient received -Tramadol/ Cremaffin

On 19/07/2014 investigation work up revealed (refer day-3 in table-1, 2a and 3):

- Drop in levels of her hemoglobin
- Raised total WBC count, platelet count, serum creatinine and blood urea nitrogen
- Total IV + oral input-5000 ml. and clear urine output-5500 ml.

In view of rising serum creatinine and urea and drop in hemoglobin level, the plan of hemodialysis and blood transfusion were maintained.

On day 4:

The general condition remained same. The plan of Hemodialysis could not be executed so far in want of proper consent due to financial constrain of the patient and her relatives.

Fifth cycle of forced diuresis continued as following:

1. 0.9 % Normal saline totals of 7 pints
2. 20 mg of Furosemide in every alternate pints of normal saline

The same supportive treatment as before was continued on day-4.

On 20/07/2014 investigation work up revealed (refer day-4 in table- 2a):

- Raised creatinine, blood urea nitrogen
- Total IV + oral input-5200 ml. and total urine output-5500 ml.

Once again it was attempted to obtain the consent for hemodialysis and blood transfusion in view of rising serum creatinine and blood urea and decreased hemoglobin level.

On day 5 and 6:

Hemodialysis could not be undertaken as consent yet to be obtained.

The general condition remained same. She was on regular full diet.

Sixth and seventh cycle of forced diuresis was repeated on day-5, 6 as on day-4.

On 21st and 22nd July 2014 investigation work up revealed (refer day-5 & 6 in table-1 and 2a):

- Drop in levels of hemoglobin
- Raised serum creatinine and blood urea nitrogen
- On day-5: Total IV + oral input-5500 ml. and clear urine output-4400 ml.
- On day-6: Total IV + oral input-5500 ml. and clear urine output-3900 ml.

On day 7:

Hemodialysis was undertaken after patient's consent for hemodialysis was obtained. Additionally patient received 0.9% Normal saline IV 6 pint of over 4- 6 hours.

On 23/07/2014 investigation work up revealed (refer day-7 in table-2a.):

- Drop in, serum creatinine, blood urea nitrogen
- Abdominal wound sutures removed, wound healed well
- Total IV + oral input- 5000 ml. and total urine output-2200 ml.
- Patient was shifted to ward

On day 8:

On 24/07/2014 she remained asymptomatic, very comfortable and was on full oral diet, without I.V. fluids, Foley's Catheter.

On day 9:

On 25/07/2014 consent for blood transfusion was obtained and one pint of blood was transfused.

On day 10:

She received yet another pint of blood before discharge on next day.

On day 11:

She was discharged.

Table-2b: Biochemical tests

Work up	Day-8	Day-9	Day-10	Day-11
Ser. Creat. mg/dl	2.6	3.1	2.1	1.4
BUN mg/dl	65	64	48	37
Na^{++} mEq/l	140		138	136
K^{++} mEq/l	3.5		3.2	3.4

Table-4: Specific tests

Investigation Workup	On Day-0
Coombs test- Direct Indirect	 Negative Negative
LDH Lu/lit	Above 1000
Bilirubin total mg/dl	4.3
Bilirubin direct mg/dl	1.0
Bilirubin indirect mg/dl	3.3
SGOT Lu/lit	80
SGPT Lu/lit	15
Alk. Phosph. Lu/lit	84
Total Protein g/dl	5.0
Albumin g/dl	2.5
Globulin g/dl	2.5
Hemoglobunuria	Positive

DISCUSSION

In ABO mismatched blood transfusion two types of acute transfusion reactions (ATR) are often described.

A. Immunological reactions – Amongst the various acute immunological reactions like hemolytic, allergic, febrile non hemolytic, anaphylaxis and TRALI, only hemolytic transfusion reaction was seen in our case in spite of the major mismatched blood transfusion.

B. Nonimmunological reactions - Amongst the various acute nonimmunological reactions like marked fever with shock, congestive heart failure, air embolism, hypocalcaemia, hypokalemia and hyperkalemia none were seen in our case.

Acute Immune Hemolytic Transfusion Reaction (AIHTR) follows the major ABO mismatch blood transfusion. Here the antigen (donor red blood cells) antibody (immunoglobulin G or M present in plasma of the recipient) react and cause the rupture of red blood cell (hemolytic reactions), the intravascular clumping of red blood cells. The widespread clumping and destruction of the recipient's red blood cells finally leads to the development of disseminated intravascular coagulation and other serious effects such as acute renal failure, cardiovascular collapse and death.

The clinical consequences of hemolytic transfusion reaction (HTR) are triggered via several pathophysiological pathways. [5-8] After intravascular RBC destruction, hemoglobin is released into the plasma which remains bound with hepatoglobin, hemopexin, and albumin which is further broken down in the reticulo-endothelial system and absorbed by phagocytosis. If this absorption capacity is exceeded, free hemoglobin passes through the glomeruli and is reabsorbed by the renal tubule. If this reabsorption capacity is also exceeded, hemoglobin can be found in the urine (hemoglobinuria). The acidic pH of the urine in the renal tubules turns this hemoglobin into acid hematin. By alkalization of urine as a part of treatment modality, the solubility of acid hematin can be increased and acid in its secretion (urine) can be reduced which in turn protects against renal failure and it should be

continued till all free hemoglobin is washed out by concomitant forced diuresis using furosemide. [9,10] Usually 50 to 150 mEq of sodium bicarbonate diluted in 1 L 0.9% NS is required to be infused at a rate of 1 to 1.5 L/hour for alkalization of the urine.

This patient received injection Furosemide and injection sodium bicarbonate in alternating pints of normal saline during first three days of critical care management. The urinary pH was maintained to 8. The urine was dark red colored, oliguric - < 600 ml. on day-0 (refer table-3) and was light red and increased to1600 ml. on day-1. On day-2 the urine was clear and was increased in quantity (diuretic phase) there onwards on days-3, 4, 5 and 6. Later the urine output became in proportion to input from day-7 onwards. The hemoglobin level was on decline during initial eight days, the lowest being 6.5% whereas highest was 10.5 % on day 11 subsequent to two pints of matching blood transfusions on 9^{th} and 10^{th} day respectively (refer table-1). Polymorphonuclear leukocytosis was observed throughout the period during hospitalization (refer table-1). Levels of serum creatinine and blood urea were on rise during early management period and was improved immediately after the hemodialysis on day-7 (refer table-2a, 2b). In our case recovery was uneventful and the urinary and blood findings returned to normal at discharge.

The reports on mismatched transfusions are frequently suppressed because of the forensic consequences which can be expected and has proven to be true, since a mismatched transfusion is usually the result of a severe and avoidable error by the physician. [4] We consider it of utmost importance to report on this recorded and successfully managed case, its course, and the medical consequences for benefit of calculation of the risk and successful treatment modality to those who may end up into such life threatening catastrophe.

The following precautions will minimize the human error.

- Well drawn protocols are to be followed.
- The duties of phlebotomist to medical technician to transfusionist are to be defined and followed strictly.
- Meticulously designed training to the transfusionist is a must as he is the last person to prevent human error and the first one to identify transfusion reaction.
- It is desirable to have a well defined transfusion setup and the team.

Ultimate future hope of eliminating this kind human error is by using artificial blood (blood replacement products such as perfluorocarbon emulsions) however, it is still being tested for its benefits, limitations and pitfalls.

CONCLUSION

Transfusion errors may be due to transfusion to a wrong patient or transfusion of a wrong blood bag. Most reported serious complications of blood transfusion are because of transfusion of mismatched blood products which are avoidable through strict clinical vigilance. Therefore, to prevent the incidences of ABO-mismatched transfusion, it is very essential to identify the patient and the blood bag individually by everyone in the blood transfusion setup (the blood transfusion team), and it must be followed very meticulously and religiously rather than struggling to manage the life threatening complication subsequently in critical care units.

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Conflicts of interest: None

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