Bombay Blood Group in a Female with Postpartum Acute Kidney Injury and Sepsis: A Case Report

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ABSTRACT

Bombay blood group is a unique blood type found mainly in the Indian subcontinent. Its prevalence is about 1 per 10,000 persons in the Indian subcontinent and 1 per 1,000,000 in Europe. It was discovered in Bombay (India) in 1952. Being asymptomatic, it often remains unidentified. Here, we report a case of an incidental discovery of this blood group in a young woman. She had a history of postpartum haemorrhage and presented with postpartum acute kidney injury and sepsis. She was severely anaemic. Blood grouping and cross--matching revealed the Bombay blood group. We faced difficulty in finding compatible blood donors for her due to the rarity of the blood group. Multiple donors were screened including family members and one of her brothers was found to have a similar blood group. With haemodialysis, broad-spectrum antibiotics, and supportive management, her condition gradually improved.

Key Words: *Blood grouping and cross-matching, Acute kidney injury, Sepsis, Bombay blood group.*

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INTRODUCTION

The classical Bombay blood group is an autosomal recessive disorder in which individuals contain anti-A, anti-B, and anti-H antibodies in the serum and lack A, B, and H antigens on the red cell surface.[1] The deficiency of the H antigen is possibly a result of a point mutation in the *fucosyl transferase* 1 (FUT1) gene with or without mutation of the *fucosyl transferase* 2 (FUT2) gene.[2] This H antigen is responsible for the formation of both A and B glycoproteins, thus constituting A and B blood groups. Its deficiency leads to a complete lack of A and B antigens. The resulting anti-H antibodies create problems in pre-transfusion testing.[3] These anti-H antibodies can activate the complement system and result in haemolysis. Here, we report a case of an incidental discovery of this blood group in a young woman who was successfully managed.

CASE REPORT

A 26-year woman presented with fever, vomiting, shortness of breath, and decreased urine output for 3 days. Five days ago, she had undergone C-section complicated with postpartum haemorrhage (PPH).

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Table I: Laboratory investigations.

Test	Result	Reference Range
Complete blood count		
Total leucocyte count	15.7 × 10^3/ul	4 – 11 × 10^3/ul
Haemoglobin	4.6 g/dl	12 – 15 g/dl
Platelets	333 × 10^3/ul	150 – 450 ×10^3/ul
Mean corpuscular volume	78 fl	80 – 100 fl
Haematocrit	15.6%	36 – 45%
Renal function tests		
Urea	184 mg/dl	15 – 40 mg/dl
Creatinine	10.3 mg/dl	0.6 – 1.3 mg/dl
Serum electrolytes		
Sodium	138 mmol/l	135 – 145 mmol/l
Potassium	5.2 mmol/l	3.5 – 5.5 mmol/l
Liver function tests		
Bilirubin	1.6 mg/dl	0.3 – 1 mg/dl
Alanine transaminase	40 U/L	7 – 56 U/L
Alkaline phosphatase	186 U/L	44 – 147 U/L
Coagulation profile		
Prothrombin time	20 sec	12 – 16 sec
International normalised ratio	1.4	0.8 – 1.2

On examination, she had severe pallor, a puffy face, and mild jaundice. Chest auscultation revealed bilateral basal fine crepitations. The rest of the clinical examination was unremarkable. Regarding laboratory investigations (Table I), her complete blood count (CBC) showed haemoglobin (Hb) of 4.6 g/dl, total leucocyte count (TLC) 15,700/ul (79% neutrophils, 17% lymphocytes, 2% eosinophils, 1% monocytes, and 1% basophils), platelets 333,000/ul, mean corpuscular volume (MCV) 78 fl, and haematocrit (HCT) 15.6%. Renal parameters showed urea of 184 mg/dl and creatinine of 10.3 mg/dl. Liver function tests showed bilirubin of 1.6 mg/dl, serum alanine transferase (ALT) 40 U/L, and alkaline phosphatase (ALP) 186 U/L. Serum electrolytes showed sodium of 138 mmol/l and potassium of 5.2 mmol/l. Prothrombin time was 20 seconds (control=14 sec) and INR was 1.4. The serum ferritin was

markedly raised (8223 ng/ml). Her ultrasound abdomen and pelvis showed normal-sized kidneys with increased echogenicity, a postpartum bulky uterus, and no retained products of conception.

One blood sample was sent to the regional blood bank for blood grouping and cross-matching which revealed the Bombay blood group. Due to the rarity of the blood group, multiple donors including family members were screened and one of her brothers was Bombay-positive. The patient was put on a broad-spectrum intravenous (IV) antibiotics and dialysis was done with one unit of packed red blood cells transfusion. With strict record of intake, output and IV antibiotics, her urine output and laboratory parameters gradually improved.

DISCUSSION

Bombay blood type is characterised by a deficiency of H antigen, typically caused by defective *FUT1* and *FUT2* genes. Therefore, the protein named fucosyl transferase, which is expressed by the *FUT* genes, is deficient. This enzyme mediates the incorporation of L-fucose into the H antigen parent chain.[4] Hence, the absence of this protein leads to a deficiency of H antigen. As H antigen on the red blood cell surface is the precursor for both A- and B- antigens, this results in the loss of these antigens. All of this is responsible for the production of anti-A, anti-B, and anti-H antibodies and the simulation of Bombay blood group with O blood group.

Bombay phenotype was initially misinterpreted as O group as it lacks red cell surface antigens similar to the O group.[5] In this blood group, agglutination of reagent O cells is seen in reverse typing but there is no agglutination in forward reaction. Individuals with this blood group can receive blood only from the Bombay blood type.[6] The arrangement of cross-match compatible blood is the biggest therapeutic challenge in these individuals.[7] The transfusion options include autologous donation, directed donations from family members and rare blood donor registries, and stored or glycerolipid blood. Unfortunately, our patient could only be transfused one unit of packed red blood cells because of the unavailability of compatible blood group donors.

The detection of the Bombay blood group requires reverse typing rather than solely relying on forward typing. The forward grouping indicates the presence or absence of A- and B-antigens on the red cell surface, whereas reverse grouping suggests the presence or absence of anti-A and anti-B in serum.[8] In a resource-limited country, with a comparatively high number of consanguineous marriages and individuals having Bombay blood type, blood grouping needs to be performed using both forward and reverse grouping rather than following the routine practice of only forward grouping with the finger prick method by voluntary blood donors' societies and numerous blood banks, so that we do not miss its detection.[9] Therefore, in patients with blood group O on forward grouping, reverse grouping with O cells must be performed for the early detection of the Bombay phenotype. Screening of the whole family should be conducted if a single member comes out to have this rare blood group.

Individuals having this blood group need multidisciplinary care, including blood management strategies and the establishment of a clear plan of action. At the national level, donor registries especially for rare blood groups must be maintained.

PATIENT'S CONSENT:
Written informed consent was obtained from the patient to publish the case.

COMPETING INTEREST:
The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:
AK: Conception of the work.
UF: Analysis and interpretation of the data.
MFA: Acquisition of the data.
MS: Drafting of the work.
All authors approved the final version of the manuscript to be published.

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