CASE REPORT OPEN ACCESS

Bombay Blood Group: An Incidental Discovery in a Family

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ABSTRACT

Bombay phenotype is an uncommon blood type. It is found in approximately 1 in 10,000 people in the Indian subcontinent and approximately 1 in 1,000,000 in Europe. The lack of the H antigen (foundation stone of the ABO blood system) on red blood cells (RBCs) and in secretions is the hallmark of this phenotype. Individuals lacking this antigen do not produce A or B antigens and appear as type O on routine blood grouping. Herein, we report an incidental discovery of a family with three members having Bombay phenotype on routine antenatal examination.

Key Words: ABO blood group system, Bombay phenotype, Cross matching.

How to cite this article: Fatimah S, Mahmood A, Sabir N, Lodhi RAK, Ghafoor T. Bombay Blood Group: An Incidental Discovery in a Family. *JCPSP Case Rep* 2023; **1**: 12-13.

INTRODUCTION

There are more than 30 blood groups and the ABO blood group is one of the most typed blood group systems in the world. The individuals lacking Hantigen contain naturally occurring Anti-Halloantibodies in their blood. When these individuals are exposed to any blood group, a severe blood transfusion reaction is observed. This is a quite rare occurrence. Bombay phenotype is usually discovered incidentally or on family screening. ^{1,2} It was initially discovered in Bombay, India in 1952 by Bhende *et al.*, and was named after the city it was discovered in. It is common in the South Asian population compared with others. It can be missed easily if blood grouping is not done by doing both forward and reverse blood grouping. Due to the infrequency and restrictions of transfusions, it is often not easy to manage individuals with Bombay groups in emergencies.

Herein, we report an incidental discovery of a family with three members having Bombay phenotype on routine antenatal examination.

CASE REPORT

A 37-year female, gravida 4 and para 3, presented to an obstetrician for delivery at the 39th week of pregnancy. She had delivered 3 daughters normally. Her routine investigations revealed low haemoglobin levels of 10.5 g/dL and considering her ultrasound findings of low lying placenta, arrangement of 2 units of packed red blood cells after cross-match was advised by her obstetrician.

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Received: January 12, 2023; Revised: March 17, 2023;

Accepted: March 28, 2023

DOI: https://doi.org/10.29271/jcpspcr.2023.12

Her workup including blood grouping and cross matching revealed O Rh D negative blood type, which was incompatible with multiple units. The initial workup was done at a peripheral hospital where her serum was treated with 11 cells antibody identification panel which revealed multiple antibodies including Lutheran Lu and Kell (k, Kp $^{\text{b}}$). Auto control was not performed in that setup.

She was then referred to our centre for a second opinion with allthe reports containing previous workup. In our setup, her forward-blood group was O Rh D negative, while the reverse groupingrevealed strong reactions with A, B, and O cells. For confirmationthe red cells when mixed with anti - H Lectin (Ulex Europeaus) showed no reaction. Both these findings (positive reaction with O cells and no reaction with anti - H Lectin) were suggestive of Bombay phenotype. We also performed auto control which wasnegative. Antibody screening and identification panel was alsorun which came out to be pan reactive, pertaining to the anti Hantibody present in Bombay Group reacting with O cells of screening and identification panel.

Considering the infrequency of this blood group phenotype, she was advised family screening which revealed her mother, sister, and one brother to be B Rh D positive, one of her two brothers Bombay Rh D positive, and her father Bombay Rh D negative. All her three daughters were B Rh D Positive like their father. She went into normal labor and delivered a normal healthy boy having blood group B Rh D positive on forward grouping, as neonates have maternal antibodies rendering their reverse grouping insignificant. No genotype was done for confirmation of the group. The patient experienced no significant haemorrhage or complication. She was not transfused blood and was discharged on the next day. At the time of discharge, her haemoglobin was 9.5 g/dL and the boy had no sign of hemolytic disease of the fetus and newborn (HDFN).

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DISCUSSION

The Bombay blood group individuals lack Hantigen due to a mutation (inactivating mutation in exon 4 of FUT1 and deletion of exon 2 in FUT2) leading to deficiency of fucosyltransferase, and production of naturally occurring anti-Halloantibodies (predominantly IgM, active at 37°C), which react with all forms of blood groups, including 0 blood group having Hantigen. They are often mislabeled as the O blood group regardless of their ABO genotype. There is no agglutination in the forward reaction but there is agglutination with both A1 and B cells in the reverse typing. To overcome this obstacle, O cell suspension is used especially in the case of group O to detect anti-Hin serum. For confirmation of diagnosis, a reaction with Ulex europaeus and molecular studies are done.

It was fortunate that our patient did not require transfusion, as the biggest therapeutic challenge in these individuals is the arrangement of cross-match compatible blood. The transfusion options for these individuals include autologous donation, directed donations from relatives, rare blood donor registries, and stored/glycerolipid blood. In the peculiar settings of pregnancy, autologous donations are not usually done as in non-pregnant settings since these can lead to maternal and fetal complications.

However, given these circumstances, individuals with Bombay phenotype require multidisciplinary care involving patient blood management strategies and the establishment of a clear plan of action. In our resource-constrained country, where there are higher percentages of consanguineous marriages and Bombay phenotype, it is essential to perform blood grouping using both forward and reverse grouping rather than relying solely on the routine practice of "only forward or cell type grouping" with finger prick method by voluntary blood donors' organisations and various blood banks, so that we do not miss its detection. As the frequency of Bombay blood group is high in our population, so for early detection, reverse grouping with O cells must be performed at least in patients with blood group O on forward grouping. In Bombay blood group patients suffering from transfusion-dependent anaemia, it is very difficult to arrange donors. At the national level, donor registries must be maintained highlighting rare blood groups. Family screening must be conducted if a single person comes out to have rare blood group. Efforts must be put together by the transfusion medicine community to start glycerolization of rare blood to preserve this asset for later use.

PATIENT'S CONSENT:

Written informed consent was obtained from the patient.

COMPETING INTEREST:

The authors declared no competing interest.

AUTHORS' CONTRIBUTION:

SF, AM, NS, RAKL, TG: Substantial contributions to the conception, drafting the work, and revising it critically for important intellectual content.

All the authors have approved the final version of the manuscript to be published.

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