Case Report

Detection of a Rare Blood Group ‘Bombay (Oh) Phenotype’ – A case report from Asgar Ali Hospital

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INTRODUCTION

The Bombay phenotype was first described by Dr. Y. M. Bhende. It was named after the city where the first case was found, Bombay (Now Mumbai). The genetic inheritance is said to be recessive with the presence of a double dose of hh gene.

The human blood group system is a group of antigens encoded by alleles at a single gene locus. H gene (FUT1), located in the long arm of chromosome 19 (19q13.3), and the Secretor (FUT2) gene are responsible for the formation of H antigen. H gene gives rise to glycosyltransferase that adds L fucose to a precursor substrate to produce H antigen. According to an individual’s ABO blood type, the H antigen is converted into either A or B antigen, or both. O blood group has inactive transferase, so H antigen remains unmodified and present in the highest amount. H antigen is present on all human red cells except rare phenotypes like Bombay and Para Bombay. Bombay phenotype has no ‘A’ or ‘B’ or ‘H’ antigens on red cells or secretions. Their serum contains naturally occurring (IgM) anti-A, anti-B and anti-H. Anti-H can activate the complement cascade and cause intravascular hemolysis. Bombay phenotype could be Rh positive or Rh negative. Para Bombay (Ah, Bh) is observed in individuals with weakly expressed A or B, but completely devoid of H antigen or a small amount of H antigen on their red cells.

People with Bombay phenotype are very small in number. A high proportion of Bombay phenotype is caused by homozygosis. The literature review revealed Bombay phenotype is more prevalent in closed-off communities where a high rate of consanguineous marriage is very common. Bombay phenotype individuals inherit the recessive form of the allele for H antigen from each of their parents. Instead of the homozygous dominant (HH) or heterozygous (Hh) genotype of the ABO blood groups, they carry the homozygous recessive (hh) genotype. As a result, the H antigen is not expressed in the red blood cell surface and subsequently A and B antigens are not formed. So it is very likely that other family members could carry the same phenotype. Thus proper screening among such families needs to be done.

During blood grouping reaction with O control cell in reverse grouping gives a clue to Bombay phenotype. Confirmation can be done by H lectin (Ulex europaeus) having anti-H like activity. Cells with blood group ‘O’ will react with it as it has highest amount H antigen and no agglutination will be seen in Bombay phenotype. Other additional specialized tests are, cross-matching with Bombay and non-Bombay O blood, and titration of naturally occurring antibodies at different temperatures. There is no ill effect of being H deficient. It is important to know the prevalence of this rare phenotype among different populations so that various measures could be taken. Here we present a case report from Bangladesh.
CASE REPORT

A 65-year-old male patient was admitted to ICU of Asgar Ali Hospital, Dhaka and diagnosed with subarachnoid hemorrhage. After admission, his blood sample was sent to the Transfusion Medicine department for routine blood grouping. Blood grouping was done by column agglutination technique (Ortho BioVue ABD forward and reverse cassettes, Ortho Clinical Diagnostics, USA). Both forward grouping including anti-A, anti-B, anti-D and reverse grouping with in-house freshly prepared pooled A cell, B cell and O cell were performed on the sample according to international standards. Patient’s blood samples with O blood group showed strong agglutination with panel O cell in a reverse grouping. The sample was tested for confirmation of Bombay blood group after excluding the presence of cold antibody.

For confirmation of Bombay blood group, the sample was tested with commercial anti-H lectin (Tulip Diagnostics, India). Tube method was used for this serological test and one drop of anti-H lectin and one drop of 5% red cell suspension were mixed, shaken to homogenize, and then centrifuged and checked for agglutination. There was no reaction and the patient was confirmed as having Bombay blood group. Indirect antiglobulin technique (IAT) cross-match with O blood group was also done which was 4+ incompatible. His rhesus phenotype was Cde / CDe (R1R1). The extended family study could not be done due nonavailability of samples from the patient’s family members. The patient needed no blood transfusion.

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Table 1: Serological reactions of Bombay patients

<table>
<thead>
<tr>
<th>Anti A</th>
<th>Anti B</th>
<th>Anti</th>
<th>A cell</th>
<th>B cell</th>
<th>O cell</th>
<th>Anti-H lectin</th>
<th>Blood group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case</td>
<td>0</td>
<td>0</td>
<td>4+</td>
<td>4+</td>
<td>4+</td>
<td>3+</td>
<td>0</td>
</tr>
</tbody>
</table>

DISCUSSION

Bombay blood group is one of the rarest blood groups found in various frequencies in different parts of the world. Suraci et al stated this H-deficit phenotype is mostly confined to Southeast Asia. The incidence is 1 in 250,000 among Caucasian. It has the highest prevalence in India and is found in 1 in every 10,000 individuals. It is believed that this blood group resulted from gene mutation in Indian population and slowly spread all over the world. The prevalence varies in various regions of India. In a study in Northern India the prevalence was found 0.0034% among blood donors. Prevalence in Southern Bengal, Tamil Nadu, Karnataka and Andhra Pradesh is 0.007%, 0.004%, 0.005% and 0.05%7. The Bombay phenotype has been reported in Japan, Malaysia, Thailand, Sri Lanka, Iran and Tehran7-11. Yunis et al reported 7 individuals with Bombay phenotype in US in an Indian family. In Bangladesh first documented Bombay phenotype was in 1990. Prevalence among Bangladeshi population was found to be 0.006%14.

Bombay phenotype can be easily misinterpreted as blood group ‘O’ if blood grouping is not correctly and thoroughly carried out. At present, in our country there is a routine practice of doing ABO and RhD typing by only ‘forward or cell type grouping’ using the finger prick method in the vast majority of cases. Rarely reverse grouping with control ‘O’ cell is done. This results in incorrect blood group typing and misinterpretation or unexploration of Bombay blood group. Catastrophic hemolytic transfusion reactions are a serious threat in such cases. So mandatory forward and reverse grouping along with ‘O’ control should be done in every Transfusion Medicine Department or Blood Bank or Blood Donor centers. In suspected cases anti-H lectin should be used for confirmation as it differs from ‘O’ blood group by lacking H antigen on RBCs.

Bombay phenotype can donate red blood cells to any member of the ABO blood group system unless some other blood factor gene, such as Rhesus is incompatible. They can receive fresh frozen plasma and cryoprecipitate from any group but they can receive red cells only either from autologous blood or from another Bombay individual. In emergencies, they should be supported with plasma or plasma expanders. It is recommended that individuals with Bombay blood group should get all their family members and relatives tested for the blood group as it is very likely that one or the other relative has this group. A Bombay phenotype individual should always be cautious and alert. They should register themselves with leading blood banks so that in case of emergency they can be contacted. Therefore, every blood bank should maintain a rare donor registry.
CONCLUSION
Being a rare blood group emphasizes the fact that proper blood grouping and cross-matching are so vital elements to ensure safe blood transfusion. The incorporation of ‘O’ control cell in the reverse grouping is very important in detecting the rare but clinically significant Bombay phenotype. This is high time to ensure a rare blood donor registry at least in tertiary care centers in Bangladesh to make a robust network for blood distribution when needed.

REFERENCES