

Transfusion reaction in a case with the rare Bombay blood group

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Abstract:

Bombay phenotype is extremely rare in Caucasian with an incidence of 1 in 250,000. When individuals with the Bombay phenotype need blood transfusion, they can receive only autologous blood or blood from another Bombay blood group. Transfusing blood group O red cells to them can cause a fatal hemolytic transfusion reaction. In this study, we report a case with the rare Bombay blood group that was misdiagnosed as the O blood group and developed a hemolytic transfusion reaction. This highlights the importance of both forward and reverse typing in ABO blood grouping and standard cross-matching and performing standard pretransfusion laboratory tests in hospital blood banks.

Key words:

ABO blood-group system, blood group incompatibility, blood transfusion, Bombay phenotype, transfusion reaction

Introduction

Bombay phenotype is one of the rarest ABO blood groups. The antigens of ABO group (A, B, and H) consist of complex carbohydrate molecules. The expression of A and B antigens is determined by the presence of H antigen on red blood cells. H antigen can be synthesized by H gene (FUT1) which is located on chromosome 19 and give rise to glycosyltransferase that add L-fucose to a precursor substance to produce H antigen on red cells. H antigen is an essential substance to A transferase or B transferase which are encoded by the ABO genes located on chromosome 9.^[1] A and B transferases convert H antigen into either A or B antigens, respectively. In group O individuals, the O allele produces an inactive transferase. Therefore, H substance persist unchanged as group O.^[2] Individuals with extremely rare Bombay phenotype fail to express H transferase. They cannot synthesize A or B antigens, and ABH antigens are absent from their red cells, regardless of their ABO blood group genotype.^[3] Since their red cells do not react with anti-A, anti-B, and anti-AB antisera, they can be recognized as the O blood group in cell typing. Their plasma contains anti-A, anti-B, and strong anti-H which can be hemolytic and is reactive with all blood types except the Bombay phenotype. As a result, individuals with the Bombay phenotype can only be safely transfused with autologous blood or other Bombay red cells.^[4]

Case Report

A 62-year-old woman was referred to our hospital because of chest pain. She was admitted to the

coronary care unit (CCU) ward with diagnosis of unstable angina. Angiography showed severe three-vessel coronary artery disease. After performing percutaneous coronary intervention, her hemoglobin level was checked and it was 90 g/L. The physician ordered one unit of packed red blood cells for her. Her blood sample was taken and sent to the hospital blood bank for performing pretransfusion tests. Her blood group was determined as the O blood group, and one unit of group O red blood cells was prepared for her. During transfusion of O red blood cells, she developed nausea, restlessness, back pain, hypotension, fever, and chills. Transfusion was immediately stopped and with a presumption of transfusion reaction, her before-transfusion blood sample was rechecked for blood grouping and cross-matching. During cell typing (forward typing), it did not show any reaction to anti-A and anti-B antibodies just like a normal O blood group. When cross-matching with the blood bag of group O was done, it showed incompatibility. A direct antiglobulin test on the after-transfusion blood sample was positive. Her blood sample was sent to the local blood transfusion center for detecting the cause of mismatching. Standard cell typing, reverse typing with O group control cells, and antibody screening and identification were performed. There was a discrepancy between cell typing and reverse typing. In reverse typing, her serum showed strong agglutination with O group control cells. The results of antibody identification showed the presence of a strong antibody which reacted with all panel cells through a wide thermal range with a negative autocontrol. Her red blood cells tested with anti-H antibody, but there was not any reaction to anti-H.

Access this article online

Website: www.ajts.org

DOI: 10.4103/0973-6247.106754

Quick Response Code:



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The probability that the patient was carrying the rare Bombay blood group raised and further confirmation of the Bombay blood group was done by a reference serology laboratory of Iranian blood transfusion organization. Since the hemoglobin level of the patient decreased to 68 g/L after reaction, two units of the Bombay blood group were sent to Yazd from the blood bank of rare blood groups which was established by Iranian Blood Transfusion Organization for such patients. These two units were cross-matched using the patient's serum and found to be compatible. After transfusing two units of Bombay blood, the patient's hemoglobin improved to 100 g/L and she was referred to a specialized cardiac surgery center for CAD management.

Discussion

The Bombay phenotype was first explained by Bhende *et al.* in 1952 in India.^[5] They showed the incompatibility of a case with the O blood group with several group O donors. The prevalence of Bombay phenotype is 1:10,000 in India and 1:10⁶ in Europe. It is also rare in Caucasian with an incidence of one in 250,000.^[6,7] In Iran, although more than 20 cases were recorded by Iranian Blood Transfusion Organization, most of the reported cases were either blood donors or hospital cases looking for blood transfusion.^[8] Therefore, the accurate prevalence of the Bombay phenotype, which is founded on random population studies, is not exactly known in Iran. However, further cases of the Bombay phenotype may become more prevalent because of immigration, consanguineous marriages, and global redistribution.

Since individuals with the Bombay phenotype are easily misdiagnosed as the O blood group in cell typing and because of the presence of strong anti-H in their plasma, if they receive blood group O red cells or any other blood group red cells except the Bombay group, they may develop an acute hemolytic transfusion reaction. This reaction can cause acute renal failure or disseminated intravascular coagulation (DIC) which is associated with high morbidity and mortality rates especially in unconscious patients who may receive large volumes of incompatible blood before signs of hemolytic reaction appears.^[9,10] The case of this study developed symptoms and signs of transfusion reaction only a few minutes after starting blood transfusion and transfusion was immediately stopped. Therefore, laboratory investigations did not demonstrate development of renal failure or DIC after reaction. However, in the hospital blood bank only slide method cell typing had been done and there was no documentation for performing standard compatibility tests.

In Iran, only forward or cell type blood grouping is performed routinely using the slide method by various hospital blood banks

and there is not any appropriate blood bank documentations for performing standard cross-matching; therefore the probability of misinterpretation of the Bombay blood group is very high. When misdiagnosed, this Bombay group can cause a hemolytic transfusion reaction. Recently, programs for implementation of the standard tube method cell typing have been started through establishment of the hemovigilance system in selected hospitals in Iran, but it is not enough. Our suggestion is to add serum typing or reverse blood grouping along with O control cells in reverse blood typing and also antibody screening in every blood bank and this practice should be obligatory to decrease the risk of hemolytic transfusion reactions. Also we recommend use of O control cells in reverse blood typing in blood transfusion centers to identify blood donors with Bombay phenotypes.

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Cite this article as: Shahshahani HJ, Vahidfar MR, Khodaie SA. Transfusion reaction in a case with the rare Bombay blood group. *Asian J Transfus Sci* 2013;7:86-7.

Source of Support: Nil, **Conflict of Interest:** None declared.