www.jcimcr.org

JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 2

Patient with rare blood group B(A) phenotype: A case report

Avineesh Chandra[1]; Bala Bhasker[2*]; Romesh Jain[3]; Rakesh Kumar[4]; Subodh Srivastava[5]

[1]Department of Blood Bank, Regency Hospital Ltd, Kanpur, UP, India.

[2]Department of Transfusion Medicine, Sparsh Multi Specialty Hospital, Bangalore, India.

[3]Department of Transfusion Medicine, AIIMS, Bhopal, India.

[4]Department of Blood Bank, Regency Hospital Ltd, Kanpur, UP, India.

[5]Department of Blood Bank, Regency Hospital Ltd, Kanpur, UP, India.

***Corresponding Author: Bala Bhasker**

Department of Transfusion Medicine, Sparsh Multi Specialty Hospital, Bangalore, India.

Email: balag4g@gmail.com

Received: Nov 08, 2021

Accepted: Dec 22, 2021

Published: Dec 29, 2021

Archived: www.jcimcr.org

DOI: DOI: www.doi.org/10.52768/2766-7820/1527

Introduction

The ABO blood group system plays a crucial role in transfusion and transplantation practices [1]. Correct identification of ABO blood group in blood donors and transfusion recipients is paramount importance for compatible transfusion of red blood cells because of minor errors can lead to fatal transfusion reactions [2,3]. Serological methods are routinely used to determine the antigens of ABO blood group system and iso-agglutinin in serum. Weak expression of A, B and H antigens on the red cell surface are often inherited or acquired. They are probably the least frequently encountered and pose a challenge in the routine immune-hematology practice [4]. In B(A) phenotype, an autosomal dominant phenotype, there is a weak A expression on group B red blood cells [5]. So, it's usually detected as an ABO discrepancy during blood group confirmation. It could be missed if only the cell grouping is performed and may lead to mislabeling of the patient/recipient blood group. This may cause hemolysis in B(A) phenotype patient if patient is transfused with A or AB Packed Red Blood Cells (PRBCs) or might end up in cross match incompatibilities.

Therefore, some ABO subgroups require molecular method to identify and also to correlate the change between DNA sequence variation in the ABO gene and the quality and quantity of blood group antigens on RBCs [6]. Here we would like to share a case of probable B(A) phenotype in a patient who was diagnosed as chronic liver disease with acute pancreatitis. To the simplest of our knowledge, this is the primary report of probable B(A) phenotype in a patient from India.

Case report

A 65-year-old female was admitted to our tertiary care center with one-week history of pain in abdomen, loss of appetite, generalized weakness. A complete blood count revealed Hb: 7.8 g/dL. Patient diagnosis was Chronic Liver Disease (CLD) associated with pancreatitis. Blood requisition was received at our blood bank for two-units of packed red blood cells (PRBC) in view of anemia. Patient blood grouping was performed using fully automated immune-hematology analyzer (MatrixAutoMax-80, Tulip Diagnostics (P) Ltd, India) by cell and serum grouping on gel column agglutination technology. For

cell grouping, the 3 micro-tubes used contains Monoclonal Anti-A, Anti-B and Anti-D anti-sera in each well and for serum grouping, the 3 other micro-tubes used contains pooled A, B and O cells in each well. The test result was reported as "Test Invalid" due to the Blood group discrepancy observed in cell and serum grouping. In cell grouping, patient red cells were reactive (2+) with anti-A antisera and strong (4+) agglutination with both anti-B and anti-D antisera where as in serum grouping, 4+ agglutination was found in micro-tube containing A pooled cells with no agglutination in micro-tubes containing B and O pooled cells.

Repeat blood grouping was done by the Conventional Tube Technique (CTT) with another fresh sample using commercially available monoclonal anti-sera manufactured by Tulip Diagnostics (P) Ltd, India. The results were corroborating with the previous results. The results of Cell grouping when performed at Room Temperature (RT) showed 2+ agglutination with anti-A antisera and strong agglutination (4+) with Anti-B, Anti-AB, Anti-D antisera and 3+ agglutination with anti-H antisera. There was no agglutination observed with anti-A1 lectin. In serum grouping, there was 4+ agglutination with A1 cells, 3+ agglutination with A2 cells and no agglutination with B cells, O cells and also in auto control. There was no enhancement in grades of reaction when performed at 4°C, 37°C, Anti-Human Globulin (AHG) phase and even after prolonged cold incubation. All these serological findings lead to a possibility of 'A_{weak} B' RhD positive phenotype with anti-A1 antibody. The critical value for Anti-A IgM titre determined by CTT was found to be as 64 using A1 cells.

To check for the weak subgroup of A or rule out of possibility of A antigen, cold adsorption and heat elution was performed as described in the American Association of Blood Banks (AABB) technical manual [7]. Adsorption of patient red cells was done at 4°C for 45 mins with human polyclonal anti-A serum collected from three group B donor samples. Heat elution was performed at 56°C for 10 mins. The eluate and the 'last wash' were tested in parallel with three different A, B and O donor red cells at Room Temperature (RT), 4°C, 37°C and in AHG phase. Results for both eluate and last wash were negative to all the donor cells used. Hence, the likelihood of weak subgroup of A was unlikely. Further, the Direct Antiglobulin Test (DAT), Autologous control and antibody screen were also negative using gel technique. Thus, the presence of anti-A antibody in serum grouping was corroborative to the group B antigen on donor RBCs. In view of the above serological findings, we concluded it could be a case of probable B(A) phenotype.

Discussion

The ABO gene is located at long arm of chromosome 9 (9q34) with 7 exons. Majority of the exonic DNA encoding the enzyme's entire catalytic domain of the ABO glycosyltransferases are harbored on exon 6 and 7. Studies showed that mutations of ABO gene affected A and B glycosyltransferase activity and caused variant ABO phenotypes [8]. The different B(A) phenotype occurs due to specific mutations on exon 6of B gene that cause the B transferase to synthesize small amounts of A antigen [9]. However, this mutation can occur on both exon 6 and exon 7, with exchange in amino acid of DNA [10].

In the present case, patient had no documented previous blood group and no history of previous transfusion. Family history revealed husband and children's blood group as A Rh D positive without any discrepancies. Patient cell and serum grouping revealed Type II blood group discrepancy. After serological workup, her blood group was interpreted as probable B(A) phenotype and got transfused with 2 units of cross match compatible O group red blood cells. Both the units were transfused successfully without any adverse events. Her post transfusion hemoglobin was 9gm/dl but got expired after 4-day stay in ICU due to worsened clinical condition. For further confirmation, saliva testing was not done due to unavailability of sufficient sample; molecular analysis and serum B-glycosyltransferase level estimation were not done due to lack of facilities.

This report highlights the importance of performing both cell and serum grouping for all the patient samples. Performing cell grouping alone, usually done at remote or district level blood centers will miss most of the weaker sub groups. This wrong practice may lead to mistyping of patients, cross match incompatibilities and delay in finding compatible blood unit. To the best of our knowledge, this is the first case reported in patients with probable B(A) phenotype from India. A Blood donor with similar blood group was reported from India by Jain et al. [11].

Conclusion

Weaker ABO blood group variants showing blood group discrepancies are often missed in routine practices. Advanced techniques like Adsorption-Elution, saliva secretor studies and molecular testing are required for accurate blood grouping, especially in patients needing blood and its components. This case report emphasizes on resolving blood group discrepancies in patient samples in order to providebest suitable blood components timely.

References

1. Heal JM, Liesveld JL, Phillips GL, Blumberg N. What would Karl Landsteiner do? The ABO blood group and stem cell transplantation. Bone Marrow Transplant. 2005; 36: 747–755.
2. Franchini M, Liumbruno GM. ABO blood group: Old dogma, new perspectives. ClinChem Lab Med. 2013; 51: 1545–1553.
3. Daniels G, Reid ME. Blood groups: The past 50 years. Transfusion 2010; 50: 281–289.
4. Harmening DM. The ABO blood group system. In: Modern blood banking and transfusion practices. 7th ed. Philadelphia: FA Davis Company Publications. 2019; p. 119–145.
5. Cooling L. ABO H, and Lewis Blood Groups and Structurally Related Antigens. In: Fung MK, Grossman BJ, Westhoff CM, editors. Technical Manual. 18th ed. Bethesda MD. AABB Press. 2014; p. 296.
6. Olsson ML, Irshaid NM, Hosseini MB, Hellberg A, Moulds H, Sareneva H. et.al. Genomic analysis of clinical samples with serological ABO blood grouping discrepancies: Identification of 15 novel A and B subgroup alleles. Blood. 2001; 98: 1585–1593.
7. Roback JD, Grossman BJ, Harris T, Hillyer CD, editors. Technical manual – American Association of Blood Banks. 20th ed. Bethesda: AABB Press. 2029; p. 880–881.

8. Daniels G. Human blood groups. 3rd. Oxford, UK: Wiley-Blackwell. 2013; p. 11–95.

9. Guo ZH, Xiang D, Zhu ZY, Liu X, Chen HP, Wang JL et. al. Serologic and molecular characterization of the B(A) blood group in the Chinese population. Immunohematology. 2007; 23: 69–74.

10. Chen Q, Li J, Xiao J, Du L, Li M, Yao G, et al. Molecular genetic analysis and structure model of a rare B(A) 0_2 subgroup of the ABO blood group system. Transfus Apher Sci. 2014; 51: 203-208.

11. Jain A, Gupta A, Malhotra S, Marwaha N, Sharma RR, et al. An ABO blood grouping discrepancy: Probable B(A) phenotype. Transfus Apher Sci. 2017; 56: 459-460.