Original Research Paper

Volume - 15 | Issue - 03 | March - 2025 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Immunohematology

CLINICALLY SIGNIFICANT ANTI M ANTIBODY SHOWING DOSAGE PHENOMENON – A CASE REPORT

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ABSTRACT Anti-M antibodies are usually of IgM, appear as cold agglutinins and are clinically insignificant. Here we are reporting one case of anti-M showing dosage and causing incompatibility in AHG phase of crossmatching. These antibodies can be clinically significant when detected in AHG phase and can cause delayed haemolytic transfusion reaction.

KEYWORDS :

INTRODUCTION

The MNS blood group system is an intricate system comprising 49 antigens. The discovery of the M and N antigens by Landsteiner and Levine in 1927 marked the beginning of understanding this complex system.[1] Later, in 1947, Walsh, Montgomery, Sanger, and Race identified the S and s antigens. Notably, the genes controlling MN and Ss antigens are closely linked, with M and N alleles, as well as S and s genes, exhibiting codominance. Interestingly, antibodies against M and N antigens are predominantly naturally occurring.[2]

Anti-M, a relatively common naturally occurring antibody, primarily of the IgM class, reacts optimally at 4°C and exhibits a characteristic antigen dosage effect. This means that anti-M reacts more strongly with M+N- red cells than with M+N+ red cells, which can complicate antibody identification. Although typically clinically insignificant, anti-M can occasionally have profound clinical implications when reactive at 37°C and can cause haemolytic disease of the newborn (HDN) and delayed haemolytic transfusion reactions (HTR) caused by rare IgG anti-M. [3,4]

This case report highlights the detection of anti-M during pretransfusion compatibility testing, which led to discrepancies and incompatibilities in cross-matching due to the dosage phenomenon.

Case Report

A 32-year-old female patient with G2P1L1 was admitted in Obstetrics and Gynaecology for delivery. Her hemoglobin was 9.0 g/dl and hematocrit 29 %. Request for arranging PRBCs was received in the blood centre. She had no H/o abortion, blood transfusion and any other clinically significant history.

The Blood Group of the patient was typed as B Rh (D) Positive by column agglutination technology. On crossmatching few red blood cell units were incompatible by column agglutination technology (Diamed ID Microtyping System).

A complete immunohematological workup of the case was initiated. Direct antiglobulin test (DAT) was performed on red cells from EDTA sample using polyspecific antiglobulin reagents (anti IgG and C3d) and was found to be negative along with negative autocontrol. Indirect antiglobulin (IAT) test using pooled O positive cells was also positive.Antibody screening with commercially available three cell panel (ID-DiaCell I-II-III Asia), showed agglutination with panel II and negative reaction with panel I and III cells (Fig. 1). Subsequently, antibody identification using 11 cell panel (ID-Diapanel) was carried out and anti-M antibody detected. It showed 3+ reaction with homozygous cells panel 1,6,10(M+N−) and no reaction was seen with heterozygous panel cells (M+N+) (Fig. 2).

No reaction was seen when enzyme treated cells were used. To determine the immunoglobulin class of the antibody, serum was treated dithiothreitol (DTT). The antibody persisted after the DTT treatment, indicating presence of IgG component along with IgM.
Red cell phenotyping of the patient was performed and was negative for M antigen. Random bags were crossmatched with the patient's sample .Out of the 4 AHG crossmatch compatible units, 2 units were found to be M antigen negative (M–N+) and two were found to be heterozygous (M+N+).

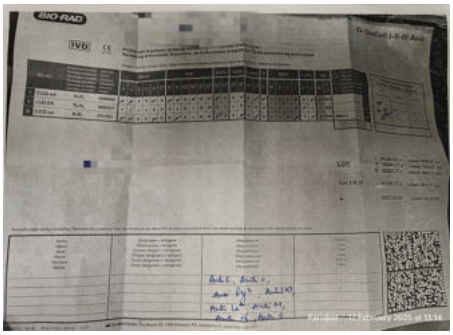

Figure 1. 3 cell Antigram showing antibody screen

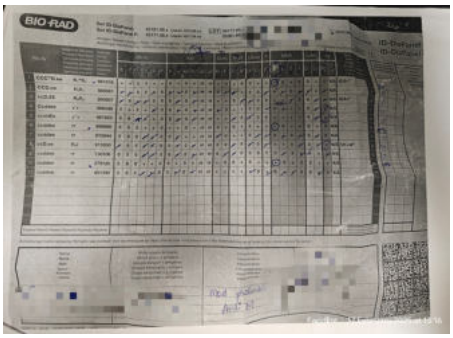

Figure 2. 11 Cell Antibody identification.

Anti-M shows dosage effect and the same was also seen in this case. Reaction was seen with units homozygous for M antigen in panel II (3 cell) and panel 1,6,10 (11 cell) and no reaction were seen when M antigen was present in single dose M+N+ (Heterozygous) in panel I and III (3 cell) and panel 3,5,8(11 cell). Even though the heterozygous units were AHG crossmatch compatible, they were not used for transfusion as they could lead to a delayed haemolytic transfusion reaction. Only one M antigen negative (M–N+) units was used for transfusion. No immediate or delayed transfusion reaction occurred.

DISCUSSION

Typically, anti-M antibodies are IgM-class, cold-reacting agglutinins that are generally considered clinically insignificant. However, in rare instances, IgG anti-M antibodies have been linked to severe complications, including delayed hemolytic transfusion reactions and

hemolytic disease of the newborn.[5]

Identifying anti-M antibodies in pretransfusion testing poses significant challenges due to the dosage effect. When reactive at 37°C, anti-M may cause incompatible cross-matches or false compatibility due to the dosage phenomenon. This occurs when donor RBCs with the M antigen (M+N+) fail to react optimally with the patient's anti-M serum.[6]

Anti-M reacts more strongly when M antigen is present in double dose M+N− (homozygous cells) and may not react with single dose M+N+ (heterozygous cells) as seen in our case report. To ensure compatibility, patients with reactive anti-M at 37°C should receive antigen-negative red blood cells.[7]

Routine pretransfusion testing should include antibody screening for all samples, followed by AHG cross-matching to detect such cases and prevent potential transfusion reactions.[8]

CONCLUSION

While most anti-M antibodies are of the IgM type, our serological analysis revealed that some are also of the IgG type, which are reactive at 37°C in the AHG phase, rendering them clinically significant. Therefore, to ensure safe transfusions, patients with anti-M antibodies should receive M-antigen negative packed red blood cells (PRBCs).

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