Hemolytic Transfusion Reaction due to Anti-JK[a]

Swathandran Hamsavardhini[1], P.Arumugam[2]

[1,2]Department of Transfusion Medicine, the Tamil Nadu Dr.M.G.R.Medical University, Guindy, Chennai-600 032, Tamilnadu, India

Abstract: *Kidd system antibodies occur only as immune antibodies and are characteristically difficult to detect. They show variability in immunoglobulin class, sub-class and serological characteristic. Kidd antibodies are dangerous as they may cause severe acute hemolytic transfusion reaction and delayed hemolytic transfusion reaction. A 19 year old male, case of carcinoma prostate with the history of previous red cell (RBC) transfusions was admitted to hospital for radiotherapy with severe anemia. On admission hemoglobin (Hb) level was 5.5 gm and hematocrit (Ht) 18%. The blood sample of the patient was sent to laboratory for serologic testing since RBC transfusions were required. Patient appeared to be A Rh(D), and one unit of packed RBCs with negative cross-matching (tube method) was issued. The transfusion was interrupted 2.5 hours after being started because of a transfusion reactions chills, lumbar pain, breathlessness and red color urine. Acute Haemolytic Transfusion Reaction (AHTR) was suspected and further evaluation was done to confirm the transfusion reaction. Direct Antiglobulin Test (DAT) was positive, Elute prepared and JK[a] antibody was identified by ID-Dia MedGel Technique. This study findings shows that it is important to monitor clinical effect of transfusion regularly and to provide good team work between specialists of transfusion medicine and related medical staff.*

Keywords: Kidd antibodies, transfusion reaction, DAT, AHTR

I. Introduction

The Kidd blood group discovered in 1951. Name derived from the initials of the sixth child of the antibody maker, Mrs. Kidd [1]. Antigens of the Kidd system are detected only on RBCs. and are carried by an integral membrane glycoprotein, which transports urea through the RBC's membrane. Urea transporter is expressed on RBCs and on endothelial cells of vasa recta in the kidney. The Kidd antibodies are very heterogeneous, clinically significant immune antibodies which are produced as a consequence of immunization by transfusion, pregnancy, rarely by transplantation or as a result of autoimmune process. Kidd antibodies have been frequently implicated in DHTRs but extremely rare are the cause of AHTRs. Following immunization, Kidd antibodies fall rapidly to undetectable levels in the plasma, therefore they are often difficult to detect [2].

The Kidd antibodies are mainly of the class IgG, subclasses IgG1 and IgG3, capable to bind complement. They might be the cause of the hemolytic disease of the fetus and newborn (HDN) but rarely severe ones. Those antibodies may manifest a dosage effect reacting only with red cells with double dose of the antigen (homozygous cells) [3]. The Kidd antibodies react better on antiglobulin testing with polyspecific anti-IgG + anti-C3 as well as with monospecific anti-C3 since they are usually detected indirectly through the complement that they bind to RBCs, therefore the reaction with monospecific anti-IgG usually lack. These antibodies usually give stronger hemo-agglutination with enzyme treated test cells [4].

II. Patient and Methods

A 19 year old male, a case of carcinoma prostate admitted in radio oncology ward for radiotherapy whose Hb was 5.5gm, hematocrit 18%, platelet count 1.5 lakhs, blood group was A +ve and he had history of 2 units of packed red cell transfusion 1 year back. A request was sent to blood bank to issue one unit of A +ve packed red cells by the attending physician to treat anemia. He received one unit of A +ve compatible blood packed red cells was cross matched by tube technique and issued to the patient. The transfusion was interrupted 2.5 hours after being started because of a transfusion reaction: chills, lumbar pain, breathlessness and red color urine. The patient was treated with IV fluids and supportive therapy and the patient made an uneventful recovery from the reaction. The pre-transfusion and post-transfusion data indicate that there was no significant change in body temperature (pre-transfusion +37.2 °C - post-transfusion +37.5 °C) and minor modifications of blood pressure (pre-transfusion 120/ 70 - post-transfusion 100/60) and heart rate (pre-transfusion 78 beats per min - post-transfusion 88 beats per minutes).

To determine the cause of the post-transfusion haemolysis, immediately after the reaction, the patient's pre & post-transfusion serum investigations were performed. The patient pre transfusion blood and post transfusion blood samples were rechecked for blood grouping, cross matching and antibody screening to rule out clerical and technical error. Antibody screening was carried out by using gel technology. A direct

antiglobulin test (DAT) was performed with polyspecific antiglobulin coombs dia med gel card to determine the presence of antibodies on the surface of cells. DAT was positive in post transfusion sample, then an elute test was done by acidic glycine to find out the specificity of the antibodies. The test was performed in Bio Rad I D Dia cell. As per the policy of drug and cosmetic act, the blood bank should store pre and post transfusion blood samples for 7 days, the patients initial sample was available for serological phenotyping [8]. Then the sample of the patient and the blood bag of the transfused unit were used for the phenotyping.

III. Results

In this study, anti-JK^a antibody was identified in the DAT positive post transfusion sample. The RBC appeared for antigen kidd JK^a was negative. While performing the phenotyping of the RBC of the transfused unit for JK^a antigen, the unit gave strong reaction for JK^a antigen. Thus, we confirmed that the transfusion reaction was due to anti-JK^a.

IV. Discussion

Intra vascular hemolysis due to transfusion is most often associated with lytic antibody such as anti-A and anti-B. Herbert. Polesky, Thomas J. Degnan and Maria Antoinetta Villa.[4],[5],[6] in their studies reported one case each of acute hemolytic transfusion reactions due to anti Jk^a. In all the three cases the antibodies were not detected in the pre transfusion samples. Only, these antibodies could be detected in post-transfusion fresh serum samples with the help of Anti Human Globulin Test particularly with anti-compliment activity and Sensitive Techniques like Enzyme Enhanced Gel Method.[5] Mollison in his study showed much more rapid in vivo destruction of Jk^a positive cell by anti Jk^a antibody which supports our study.[7] A dosage phenomenon was clearly demonstrated with homozygous Jk^a cell panel in the antibody identification test.

The titer of the anti Jk^a quickly declines in vivo. A strong antibody identified following a transfusion reaction may be undetectable in few weeks or months.[9] This confirms in our study that the patient had history of transfusion two years back that had an anamnestic reaction and developed transfusion reaction.

V. Conclusion

In view of the fact that the concentration of anti- Jk^a declines very fast to the undetectable levels soon after allogenic immunization, it is utmost important to inform the patient about the specificity of the antibody to prevent such reaction in future. Hence, even with the currently available serological methods and the closest attention to technical performance such catastrophic transfusion reactions do occur due to poorly demonstrable circulating antibodies or yet undefined immune mechanisms.

Acknowledgements

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