

CLINICAL NOTE

STANDARDIZED LECTIN IN THE PREVENTION OF BLOOD GROUP INCOMPATIBILITY

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Abstract

Background-The two principal subgroups of A antigen are A1 and A2. About 80% of group A individuals have red blood cells (RBCs) that are agglutinated by anti-A1. At appropriate dilution, *Dolichos biflorus* (lectin) acts as an anti-A1 and agglutinates A1 but not A2 RBCs. The main goal of this study was to test and introduce a standardized lectin in order to differentiate the subgroups of A antigen in Iran.

Methods-The powder form of the plant *Dolichos biflorus* was subjected to a series of sequential operations according to current standard protocols with some modifications. The prepared reagent was used to detect A1 and A2 cells based upon the agglutination reaction. The reagent was initially used to detect incompatibility reaction in 450 transfused patients at Al- Zahra teaching hospital (Isfahan, Iran) and all the results were rechecked using a standard lectin.

Results-Five of 450 patients with blood group A or AB showed incompatibility with a high anti-A1 titer after transfusion of isogroup blood. Among them, 2 patients were of A2 and 3 were of A2B types. The same results were obtained by using standard lectin.

Conclusion-Considering the great importance of subgroup incompatibility in transfusion medicine, we recommend the use of standardized lectin for the differentiation of A antigen subgroups before transfusion of RBCs containing the A antigen.

Keywords • Lectin • anti-A1 • blood group incompatibility • *Dolichos biflorus*

Introduction

ABO system antibodies arise shortly after birth on exposure to environmental agents for which antigenic makeup is similar to the A and B antigen found on human red blood cells (RBCs). The antibodies of the ABO system are primarily IgM in nature, although some IgG and IgA antibodies may also be present. The immune form of the ABO antibodies results from exposure to incompatible RBCs or other sources of ABO antigens.¹

Most of the anti-A is of IgM type. Thus anti-A is able to agglutinate RBCs suspended in saline and activate complement with ease. It may cause rapid intravascular destruction of RBCs carrying

the A antigen. Anti-A can be functionally divided into two forms: one form which reacts with A1 but not A2 cells (anti-A1) and another form which reacts with both A1 and A2 cells (anti-A common). A1 and A2 are the two most common subgroups of A antigen. Anti-A1 is found in 1 to 8 percent of A2 individuals and 22 to 35 percent of A2B individuals. Other subgroups are very rare and are not important in transfusion medicine. A1 and A2 phenotypes are best differentiated using the anti-A1 (lectin) extracted from the seeds of the plant *Dolichos biflorus*.^{2,3}

The aim of this study was to standardize and introduce lectin as a reagent in order to differentiate the subgroups of A antigen in Iran.

Patients and Methods

Ulex and *Dolichos* seeds were purchased from

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the Indian Institute of Immunohematology. We extracted anti-H and anti-A1 from *Ulex urepeous* and *Dolichos* seeds, respectively.

Four-hundred and fifty group A or AB patients from Al-Zahra Hospital (Isfahan, Iran) were selected among recipients of isogroup blood in the year 2000 and were evaluated in terms of incompatibility using a routine agglutination test.

All agglutinated samples from 1+ to 4+ were considered positive. The level of anti-A1 antibody was measured in those cases with incompatibility and the titer of 1/32 and more was considered significant. Confirming the presence of A2 cells in the donor's blood, anti-H (*Ulex urepeous*) was added to the samples and agglutination reaction from 2+ to 3+ was considered positive. All results were rechecked using a type of standard lectin (EY Laboratories Inc, USA)

Results

Five of 450 patients showing incompatibility had a high anti-A1 titer (1/32 or more). Of these 5 patients, 2 cases were of A2 and 3 cases were of A2B group. A significant reaction (anti-A1 titer $\geq 1/32$) was observed at 30° C and 37°C. Using standard lectin (EY Laboratories Inc, USA) as a control test reproduced the same results.

Discussion

Some events can cause unexpected or erroneous serum test results. Immunodeficient patients may not produce detectable levels of anti-A1 and anti-B1. These antibodies are absent in the serum of newborns and may be scanty in the serum from normal elderly persons. Testing with anti-H lectin from *Laburnum albinum* may be helpful in distinguishing between A1- and A2- RBCs in the first month of life; A2 cells react much stronger than A1 cells.

Abnormally high concentrations of anti-A1 and anti-B have caused prozone reactions that lead to false-negative results. Strongly reactive cold autoagglutinins, such as anti-I and anti-IH can agglutinate the RBCs of all adults including autologous cells and reagent red blood cells, at room temperature (20-24°C) with a few exceptions; agglutinin is weaker than that caused by anti-A and

anti-B.^{3,4}

Anti-A1, which is active *in vitro* at about 30°C but only dubiously active at 37°C, will bring about the destruction of a proportion of A1 cells *in vivo* when a small dose of cells is injected. Those antibodies which are only dubiously active at 37°C would almost certainly fail to produce detectable RBC destruction following the transfusion of therapeutic quantities of blood. On the other hand, in several instances in which anti-A1 has been active at 37°C, extensive destruction of A1 cells *in vivo* has been recorded.⁵

Boorman et al (1946) reported a case in which a patient of subgroup A2 was transfused with at least 7 units of A1 blood within a period of 4 days. Seven days after the last transfusion the patient became icteric and anemic and was found to have anti-A1 in her serum active at 37°C. Several other examples of the development of anti-A1 active at 37°C following a series of transfusions have been described.⁶

Considering the lesser survival of A1- RBCs transfused to A2B or A2 persons, whose sera contain anti-A1⁷, we propose to distinguish A1 and A2 subgroups in individuals with A and AB blood groups prior to blood transfusion, especially in those with previous history of transfusion reactions following isogroup blood transfusions.

References

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