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CASE REPORT

A CASE OF RARE BLOOD GROUP IN OBSTETRIC EMERGENCY

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ABSTRACT: We present a rare case of an A2B +ve blood group with obstetric hemorrhage in mild hypovolemic shock with DIC. An emergency request for blood transfusion confounded the blood bank officer as the patient's blood was seemingly incompatible with all ABO blood groups. Further investigation revealed the patient's blood group to be a rare subtype of the A group known as A2+veB. This article highlights the need to be aware of such rare subgroups, especially in tertiary referral centers, where unbooked Obstetric emergencies are encountered on a regular basis, so life-saving measures can be appropriately taken.

KEYWORDS: Rare blood group, DIC.

INTRODUCTION: Two principal subgroup of blood group A are A1 and A2. In Indian population the frequency of A2 is 0.8-3.0%, while the frequency of A2B is 0.6-1.4%[2]. 22-26% of A2B individuals can have anti A1 antibodies that react a temperature below 25 degrees and cause hemolytic transfusion reaction[1, 2.] We present a rare case of an A2B positive blood group with postpartum hemorrhage, DIC in hypovolemic shock. An emergency request for blood transfusion created confusion in determining the exact blood group of the patient. This article highlights the need to be aware of such rare blood groups and using anti A1 lectin as a standardized protocol to prevent blood group incompatibility.

CASE REPORT: A 21 years old, female, referred from MIMS, Mandya as Para 2 living 2 delivered at 3pm at the Maddur government hospital with atonic PPH with shock in DIC. On examination patient had a feeble pulse and a BP of 80 systolic with a respiratory rate of 32 cycles/min. Her uterus was well contracted with minimal bleeding through the OS with 700 ml of hematuria present.

The patient had already been managed medically for PPH and came to us on dopamine infusion drip and 2 pint PRBC of B +ve blood on flow. After stabilizing the patient, coagulation profile was found abnormal and 6pints platelets, 1 pint cryo and 4 pints FFP's was decided to be transfused.

But when blood was sent for cross matching, patient's blood group could not be ascertained. Hence FFP transfusion was withheld for the time being. At first, our blood bank could not determine whether the blood group was AB+ve or B+ve. Then after further testing A2B blood group was confirmed. Since by that time patient had come out of shock and DIC further transfusions were avoided and her anemia was corrected with iron sucrose. The patient was discharged after 10 days of hospital stay.

Table 1: Different phenotypes of ABO in different races

Blood group	O	A_1	A_2	B	A_1B	A_2B
Caucasian	44%	33%	10%	9%	3%	1%
Black people	49%	19%	8%	20%	3%	1%
Asian	43%	27%	Rare	25%	5%	Rare

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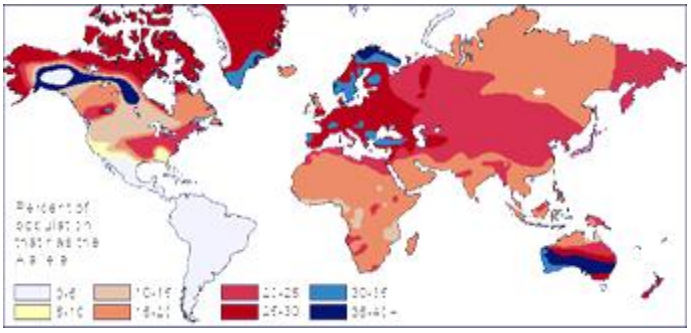

DISCUSSION AND CONCLUSION: ABO system antibodies arise shortly after birth on exposure to environmental agents for which antigenic makeup is similar to the A and B antigen found on human red blood cells (RBCs). The antibodies of the ABO system are primarily IgM in nature, although some IgG and IgA antibodies may also be present. The immune form of the ABO antibodies results from exposure to incompatible RBCs or other sources of ABO antigens. 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).[3] A1 and A2 phenotypes are best differentiated using the anti-A1 (lectin) extracted from the seeds of the plant Dolichos biflorus.[4,5]

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.

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-A1,[7] we propose to distinguish A1 and A2 subgroups in individuals with A and AB blood groups prior to blood transfusion, especially in those with a previous history of transfusion reactions following iso group blood transfusions.

Massive obstetric hemorrhage is a major contributor towards Maternal morbidity and mortality. Clinicians managing pregnant women should be equipped with the knowledge of blood and blood products and skills for managing massive obstetric hemorrhage[8]. Most of the individuals with a rare blood group are coincidently identified when a routine pre-transfusion testing or pregnancy follow-up is performed, if the antibodies corresponding to the rare specificity are present.

There is a growing awareness of the impact of the genomics revolution on transfusion medicine and its potential to transform the way blood is selected for transfusion. From antibody-based technology to now single-nucleotide polymorphism (SNP) genotyping for blood, PCR-

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technology will help in extended matching of RBC units.[9] Such advances in cross-matching of blood can save the lives of many, especially, as in this case, young women of childbearing age and thus reduce maternal mortality.

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