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Original Research Article

Frequency of ABO and Rh D Blood Groups among Sudanese Blood Donors Attending Central Blood Bank in Wad Medani, Gezira State, Sudan

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ABSTRACT

Objective: To determine the distribution of ABO and Rh D blood groups among Sudanese blood donors attending Central Blood Bank in Wad Medani and to minimize Rhesus allo-immunization among blood recipients.

Material and Methods: Five hundred male blood donors were enrolled in the study; ABO typing and Rh D were performed using the classical slide method.

Results: The study revealed that frequency percentage of ABO blood phenotypes in the total samples were as follows: O (51%), A (30%), B (14%) and AB (5%), The Majority (97.4%) of the subjects were Rh (D) positive and only 2.6% were Rh negative.

Conclusion: The frequency of ABO blood groups among the Sudanese was similar some extend with world ABO distribution with predominance of Rh D positive.

Key words: ABO Blood Group, Rh D, Blood Donors.

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INTRODUCTION

Red Cell Antigens

Since Landsteiner's discovery in 1901, that human blood groups existed, a vast body of serological, genetic and biochemical data on red cell (blood group) antigens has been accumulated. More recently, the biological functions of some of these antigens have been appreciated. A total of 30 blood group systems have been described. Each system is a series of red cell antigens, determined either by a single genetic locus or very closely linked loci. In addition to the blood group systems, there are six 'collections' of antigens (e.g. Cost), which bring together other genetically, biochemically or serologically related sets of antigens and a separate series of low frequency (e.g. Rd) and high-frequency (e.g. Vel) antigens, which do not fit into any system or collection. A numeric catalogue of red cell antigens is being maintained by an International Society of Blood Transfusion (ISBT) Working Party.[1] Apart from those of the ABO system, most of these antigens were detected by antibodies stimulated by transfusion or pregnancy. Alternative forms of a gene coding for red cell antigens at a particular locus are called alleles and individuals may inherit identical or non-identical alleles. Most blood group genes have been assigned to specific chromosomes (e.g. ABO system on chromosome 9, Rh system on chromosome 1). The term genotype is used for the sum of the inherited alleles of a particular gene (e.g. AA, AO) and most red cell genes are expressed as codominant antigens (i.e. both genes are expressed in the heterozygote). The phenotype refers to the recognizable product of the alleles and there are many racial differences in the frequencies of red cell phenotypes. Red cell antigens are determined either by carbohydrate structures or protein structures. Carbohydrate-defined antigens are indirect gene products (e.g. ABO, Lewis, P). The genes code for an intermediate product, usually an enzyme that creates the antigenic specificity by transferring sugar molecules onto the protein or lipid. Protein-defined antigens are direct gene products and the specificity is determined by the inherited amino acid sequence and/or the conformation of the protein. Proteins carrying red cell antigens are inserted into the membrane in one of three ways: single pass, multipass or linked to phosphatidylinositol (GPI-linked).

Only a few red cell antigens are erythroid-specific (Rh, LW, Kell and MNSs), the remainder being expressed in many other tissues. The structure and functions of the membrane proteins and glycoproteins carrying blood group antigens have been reviewed by Daniels.[2] However, the main clinical importance of a blood group system depends on the capacity of alloantibodies (directed against the antigens not possessed by the individual) to cause destruction of transfused red cells or to cross the placenta and give rise to haemolytic disease in the fetus or newborn.

This in turn depends on the frequency of the antigens and the alloantibodies and the characteristics of the latter: thermal range,

immunoglobulin class and ability to fix complement. On these criteria, the ABO and Rh systems are of major clinical importance. Anti-A and anti-B are naturally occurring and are capable of causing severe intravascular haemolysis after an incompatible transfusion. The RhD antigen is the most immunogenic red cell antigen after A and B, being capable of stimulating anti-D production after transfusion or pregnancy in the majority of RhD-negative individuals.

ABO SYSTEM

Discovery of the ABO system by Landsteiner marked the beginning of safe blood transfusion. The ABO antigens, although most important in relation to transfusion, are also expressed on most endothelial and epithelial membranes and are important histocompatibility antigens.[3] Transplantation of ABO-incompatible solid organs increases the potential for hyperacute graft rejection, although ABO-incompatible renal transplantation can be successfully carried out with plasmapheresis in addition to immunosuppression of the recipient.[4] Major ABO-incompatible stem cell transplants (e.g. group A stem cells into a group O recipient) will provoke haemolysis, unless the donation is depleted of red cells.

ABO ANTIGENS AND ENCODING GENES

There are four main blood groups: A, B, AB and O. In the British Caucasian population, the frequency of group A is 42%, B 9%, AB 3% and O 46%, but there is racial variation in these frequencies.[5]

The epitopes of ABO antigens are determined by carbohydrates (sugars), which are linked either to polypeptides (forming glycoproteins) or to lipids (glycolipids). The expression of ABO antigens is controlled by three separate genetic loci: ABO located on chromosome 9 and FUT1 (H) and FUT2 (Se), both of which are located on chromosome 19. The genes from each locus are inherited in pairs as Mendelian dominants. Each gene codes for a different enzyme (glycosyltransferase), which attaches specific monosaccharides onto precursor disaccharide chains. There are four types of disaccharide chains known to occur on red cells, on other tissues and in secretions. The Type 1 disaccharide chain is found in plasma and secretions and is the substrate for the FUT2 (Se) gene, whereas Types 2, 3 and 4 chains are only found on red cells and are the substrate for the FUT1 (H) gene. It is likely that the O and B genes arose by mutation of the A gene. The O gene does not encode for the production of a functional enzyme; group O individuals commonly have a deletion at nucleotide 261 (the O1 allele), which results in a frame-shift and premature termination of the translated polypeptide and the production of an enzyme with no catalytic activity. The B gene differs from A by consistent nucleotide substitutions.[6] The expression of A and B antigens is dependent on the H and Se genes, which both give rise to glycosyltransferases that add L-fucose, producing the H antigen. The presence of an A or B gene (or both) results in the production of further glycosyltransferases, which convert H substance into A and B antigens by the terminal addition of N-acetyl-D-galactosamine and D-galactose, respectively. Because the O gene produces an inactive transferase, H substance persists unchanged as group O. In the extremely rare Oh Bombay phenotype, the individual is homozygous for the h allele of FUT1 and hence cannot form the H precursor of the A and B antigen. Their red cells type as group O, but their plasma contains anti-H, in addition to anti-A, anti-B and anti-A, B, which are all active at 37º C. As a consequence, individuals with an Oh Bombay phenotype can only be safely transfused with other Oh red cells. Serologists have defined two common subgroups of the A antigen. Approximately 20% of group A and group AB Individuals belong to group A 2 and group A 2 B, respectively, the remainder belonging to group A 1 and group A 1 B. These subgroups arise as a result of inheritance of either the A 1 or A 2 alleles. The A 2 transferase is less efficient in transferring N-acetyl-D-galactosamine to available H antigen sites and cannot utilize Types 3 and 4 disaccharide chains. As a consequence, A 2 red cells have fewer A antigen sites than A 1 cells and the plasma of group A 2 and group A 2 B individuals may also contain anti-A 1 . The distinction between these subgroups can be made using the lectin Dolichos biflorus, which only reacts with A 1 cells.

The H antigen content of red cells depends on the ABO group and, when assessed by agglutination reactions with anti-H, the strength of reaction tends to be graded O > A 2 > A 2 B > B > A 1 > A 1 B. Other subgroups of A are occasionally found (e.g. A 3 , A x) that result from mutant forms of the glycosyltransferases produced by the A gene and are less efficient at transferring N-acetyl-D-galactosamine onto H substance.[6] The A, B and H antigens are detectable early in fetal life but are not fully developed on the red cells at birth. The number of antigen sites reaches 'adult' level at around 1 year of age and remains constant until old age, when a slight reduction may occur.

SECRETORS AND NON-SECRETORS

The ability to secrete A, B and H substances in water-soluble form is controlled by FUT2 (dominant allele Se). In a Caucasian population, about 80% are secretors (genotype SeSe or Sese) and 20% are non-secretors (genotype sese). Secretors have H substance in the saliva and other body fluids together with A substances, B substances or both, depending on their blood group. Only traces of these substances are present in the secretions of non-secretors, although the antigens are expressed normally on their red cells and other tissues. An individual's secretor status can be determined by testing for ABH substance in saliva.

ABO ANTIGENS AND DISEASE

Group A individuals rarely may acquire a B antigen from a bacterial infection that results in the release of a deacetylase enzyme. This converts N-acetyl-D-galactosamine into a galactosamine, which is similar to galactose, the immune dominant sugar of group B, thereby sometimes causing the red cells to appear to be group AB. In the original reported cases, five out of seven of the patients had carcinoma of the gastrointestinal tract.

Case reports attest to the danger of individuals with an acquired B antigen being transfused with AB red cells, resulting in a fatal haemolytic transfusion reaction following the production of hyperimmune anti-B.[7]

The inheritance of ABH antigens is also known to be weakly associated with predisposition to certain diseases. Group A individuals have 1.2 times the risk of developing carcinoma of the stomach than group O or B; group O individuals have 1.4 times more risk of developing peptic ulcer than non-group O individuals; and non-secretors of ABH have 1.5 times the risk of developing

peptic ulcer than secretors.[8] The ABO group also affects plasma von Wille- brand factor (VWF) and factor VIII levels; group O healthy individuals have levels around 25% lower than those of other ABO groups.[9] ABO blood group appears to mediate its effect by accelerating clearance of VWF but the mechanism is not yet clear.[10] ABH antigens are also frequently more weakly expressed on the red cells of persons with leukaemia.

ABO ANTIBODIES

Anti-A and anti-B

ABO antibodies, in the absence of the corresponding antigens, appear during the first few months after birth, probably as a result of exposure to ABH antigen-like substance sin the diet or the environment (i.e. they are 'naturally occurring'). This allows for reverse (serum/plasma) grouping as a means of confirming the red cell phenotype. The antibodies are a potential cause of dangerous haemolytic transfusion reactions if transfusions are given without regard to ABO compatibility. Anti-A and anti-B are always, to some extent, immunoglobulin M (IgM). Although they react best at low temperatures, they are nevertheless potentially lytic at 37º C. Hyperimmune anti-A and anti-B occur less frequently, usually in response to transfusion or pregnancy, but they may also be formed following the injection of some toxoids and vaccines. They are predominantly of IgG class and are usually produced by group O and sometimes by group A 2 individuals. Hyperimmune IgG anti-A and/or anti-B from group O or group A 2 mothers may cross the placenta and cause haemolytic disease of the newborn (HDN). These antibodies react over a wide thermal range and are more effective haemolysins than the naturally occurring antibodies.

Group O donors should always be screened for high-titre anti-A and anti-B antibodies, which may cause haemolysis when group O platelets or plasma are transfused to recipients with A and B phenotypes. Plasma-containing blood components from such high titre universal donors should be reserved for group O recipients.

Anti-A 1 and anti-H

Anti-A 1 reacts only with A 1 and A 1 B cells and is occasionally found in the serum of group A 2 individuals (1–8%) and not uncommonly in the serum of group A 2 B subjects (25–50%). However, anti-A 1 normally acts as a cold agglutinin and is very rarely reactive at 37º C, when it is only capable of limited red cell destruction.

There have been a few reports of red cell haemolysis as cribbed to anti-A 1, which some authors have questioned because, although the antibodies reacted only with A 1 red cells, no attempts were made to absorb them with A 2 cells, which would have revealed their anti-A specificity. Anti-H reacts most strongly with group O and A 2 red cells and also normally acts as a cold agglutinin. A notable, but rare, exception is the anti-H that occurs in the Oh Bombay phenotype, which is an IgM antibody and causes lysis at 37º C so that Oh Bombay phenotype blood would be required for transfusion.

Rh SYSTEM

The Rh system, formerly known as the Rhesus system, was so named because the original antibody that was raised by injecting red cells of rhesus monkeys into rabbits and guinea pigs reacted with most human red cells. Although the original antibody (now called anti-LW) was subsequently shown to be different from anti-D, the Rh terminology has been retained for the human blood group system. The clinical importance of this system is that individuals who are D negative are often stimulated to make anti-D if transfused with D-positive blood or, in the case of pregnant women, if exposed to D-positive fetal red cells that have crossed the placenta.

Rh ANTIGENS AND ENCODING GENES

This is a very complex system. At its simplest, it is convenient to classify individuals as D positive or D negative, depending on the presence of the D antigen. This is largely a preventive measure, to avoid transfusing a D-negative recipient with the cells expressing the D antigen, which is the most immunogenic red cell antigen after A and B.

At a more comprehensive level, it is convenient to consider the Rh system as a gene complex that gives rise to various combinations of three alternative antigens– C orc, D or d and E ore – as originally suggested by Fisher. The d gene was thought to be amorphic without any corresponding antigen on the red cell. Subsequently it was confirmed that the RH locus is on chromosome 1 and comprises two highly homologous, very closely linked genes, RHD and RHCE, each with 10 exons. Each gene codes for a separate transmembrane protein with 417 residues and 12 putative transmembrane domains. The D and CE proteins differ at 35 residues. The RHCE gene has four main alleles; CE, Ce, ce and cE. Positions 103 and 226 on the CE polypeptide, situated in the external loops, determine the C/c (serine/proline) and E/e (proline/ala-nine) polymorphisms, respectively. This concept of D and Cc Ee genes linked closely and transmitted together is consistent with the Fisher nomenclature.

In Caucasian, D-negative individuals, the RHD gene is deleted, whereas in Black races and other populations, single-point mutations, partial deletions or recombinations have been described. In individuals with a weak D antigen (D u), there is a quantitative reduction in D antigen sites, believed to arise from an uncharacterized transcriptional defect. These individuals do not make anti-D antibodies following a D antigen challenge. Partial D individuals lack one or more epitopes of the D antigen, defined using panels of monoclonal reagents. D^{VI} is perhaps the most important partial D phenotype because such individuals not infrequently make anti-D. Partial D phenotypes arise from DNA exchanges between RHD and RHCE genes and from other rearrangements.

Comprehensive reviews of this system have been provided by Avent and Reid[11] and Daniels et al.[12] The Rh haplotypes are named either by the component antigens (e.g. CDe, cde) or by a single shorthand symbol (e.g. R 1 ¼ CDe, r ¼ cde). Thus, a person may inherit CDe (R 1) from one parent and cde (r) from the other and have the genotype CDe/cde (R 1 r). Although two other nomenclatures are also used to describe the Rh system, namely, Wiener's Rh-Hr terminology and Rosenfield's numeric notation, the CDE nomenclature, derived from Fisher's original theory, is recommended by a World Health Organization· Expert Committee[13] in the interest of simplicity and uniformity.

The Rh antigens are defined by corresponding antisera, with the exception of 'anti-d', which does not exist. Consequently, the distinction between homozygous DD and the heterozygous Dd cannot be made by direct serological testing but may be resolved

by informative family studies. It is still routine practice to predict the genotype from the phenotype on the basis of probability tables for the various Rh genotypes in the population. However, in women with immune anti-D and a history of an infant affected by HDN, RH DNA typing is used in prenatal testing for the fetal D status to decide on the clinical management of the pregnancy, e.g. the need for monitoring for fetal anaemia using middle cerebral artery Doppler ultrasound. Suitable sources include amniotic fluid (amniocytes) and trophoblastic cells (chorionic villi) or after 15 weeks' gestation, maternal blood can be used because it contains fetal DNA.[14,15]

In practice, multiplex polymerase chain reaction (PCR) is used, with more than two primer sets, to detect the different molecular bases for D-negative phenotypes in non-Caucasians. RH DNA typing also has applications in paternity testing and forensic medicine. There are racial differences in the distribution of Rh antigens, e.g. D negativity is more common in Caucasians (approximately 15%), whereas R o (cDe) is found in approximately 48% of Black Americans but is uncommon (approximately 2%) in Caucasians.

The Rh antigens are present only on red cells and are a structural part of the cell membrane. Complete absence of Rh antigens (Rh-null phenotype) may be associated with a congenital haemolytic anaemia with spherocytes and stomatocytes in the blood film, increased osmotic fragility and increased cation transport. This phenotype arises either as a result of homozygosity for silent alleles at the RH locus (the amorph type) or more commonly by homozygosity for an autosomal suppressor gene (X), genetically independent of the RH locus (the regulator type). Rh antigens are well-developed before birth and can be demonstrated on the red cells of very early fetuses.

ANTIBODIES

Fisher's nomenclature is convenient when applied to Rh antibodies, and antibodies directed against all Rh antigens, except d, have been described: anti-D, anti-C, anti-c, anti-E and anti-e. Rh antigens are restricted to red cells and Rh antibodies result from previous alloimmunization by previous pregnancy or transfusion, except for some naturally occurring forms of anti-E and anti-C W. Immune Rh anti- bodies are predominantly IgG (IgG1 and/or IgG3), but may have an IgM component.

They react optimally at 37º C, they do not bind complement and their detection is often enhanced by the use of enzyme-treated red cells. Haemolysis, when it occurs, is therefore extravascular and predominantly in the spleen. Anti-D is clinically the most important antibody; it may cause haemolytic transfusion reactions and was a common cause of fetal death resulting from haemolytic disease of the newborn before the introduction of anti-D prophylaxis. Anti-D is accompanied by anti-C in 30% of cases and anti-E in 2% cases. Primary immunization following a transfusion of D positive cells becomes apparent within 2–5months, but it may not be detectable following exposure to a small dose of D-positive cells in pregnancy. However, a second exposure to D-positive cells in a subsequent pregnancy will provoke a prompt anamnestic or secondary immune response.

Of the non-D Rh antibodies, anti-c is most commonly found and can also give rise to severe haemolytic disease of the fetus and newborn. Anti-E is less common, whereas anti-C is rare in the absence of anti-D.

BASIC CRITERIA OF BLOOD DONORS AND MEDICAL EVALUATION

In addition to a medical history, donors undergo a brief physical examination before donation to check for any obvious signs of illness or conditions that would disqualify them from blood donation.

1. The donor appears to be in a good health.
2. Age 18 to 60 years.
3. Body weight 50 Kg (110 pounds) and above.
4. Temperature- not to exceed 37C.
5. Pulse shall reveal no pathological cardiac abnormalities and should be regular 50-100 beats/ minute.
6. Blood pressure: systolic pressure not higher than 180 mmHg and diastolic pressure not higher than 100 mmHg.

LABORATORY TESTING OF DONATED BLOOD

Blood test: Blood sample is taken and tested to check for the amount of haemoglobin in the blood. This is done to ensure that the donor is not anaemic or likely to become anaemic or iron deficient after they donate. Individuals with haemoglobin levels that are too low are temporarily not permitted to donate blood. Platelets count and white blood count are also important.

The haemoglobin level must be more than 12.5 g/dl with platelet count 150-400 x 10^9 per litre and normal count of white blood cells.[16]

ABO AND Rh (D)

The donor's blood group must be determined, whether it is of group A, B, AB, or O and the donor's Rh (D) type. The ABO group is tested by forward typing of donor cells using anti- A and anti- B reagents and reverse group typing of the donor sera using known A and B reagents red blood cells. Antibody screening is also performed to detect unexpected antibodies with clincal significance. Any discrepancies between forward and reverse grouping must be resolved before the donor unit can be released. Direct typing for the Rh (D) antigen is performed using an anti-D reagent with the appropriate control. Persons typing D- negative on intial testing must be examined by the antihumanglobulin (AHG) test, to detected the D^U varient (weak D) all the ABO and Rh (D) negative units must be confirmed by the hospital transfusion service prior to tranfusion.[16]

ATYPICAL ANTIBODY SCREENING

About 4 in 1000 blood donations demonstrates unexpected antibodies. All patients and donors serum or plasma should be tested for unexpected antibodies that are clinically significant other than anti-A and anti-B. This is facilitate the selection of suitable blood for the patients requiring transfusion. This test is performed using screening red cells which are available commercially. More testing, including a crossmatch, is usually done before a transfusion.[17]

METHODS

Sample collection

A total of 500 apparently healthy adult male donors were screened for ABO blood groups. This analysis was conducted at the Wad medanni central blood bank, department of pathology (medical laboratory) and the central laboratory of Wad medanni teaching hospital.

Laboratory Procedures

The blood samples were collected by finger prick with sterile lancet and after warming and cleaning the puncture site with 70% ethyl alcohol using classical slide method. A drop of monoclonal anti-A, anti-B, monoclonal /polyclonal anti-D.

Interpretation of Results

Results of agglutination were recorded immediately for ABO blood groups and after 2 minutes in Rh(D). Mixed well over an area of 2.5 cm. Slide was rocked gently back and forth. Agglutination was observed within 2 minutes, it was indicated to the presence of corresponding antigen. No agglutination is a negative result and indicated the absence of corresponding antigen.

Data Analysis

All data collection from practical and questionnaires survey was entered in Microsoft office excel. Then the result analyzed by Statistical Package for Social Sciences (SPSS) program version 20, across tab correlation was done. Statistical analysis: The results were analyzed using statistical software package of social sciences (SPSS) version 17 and descriptive data were expressed as means.

Ethical Clearance

Ethical clearance was obtained from the University of Gezira ethical committee and blood bank authority. Verbal informed consent was obtained from all donors.

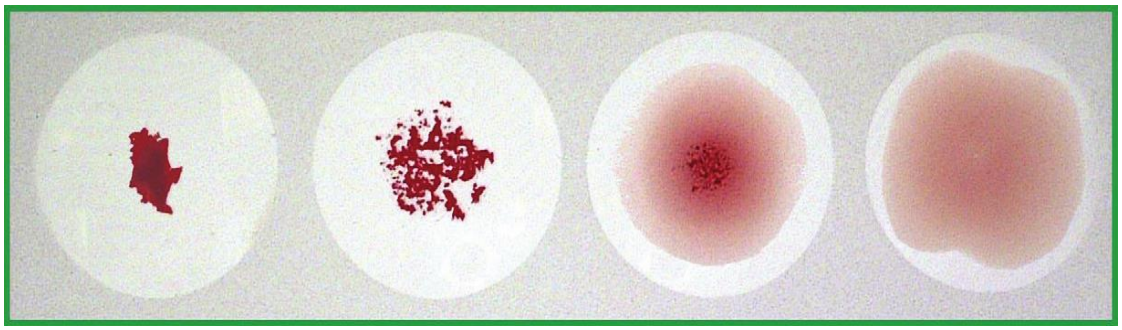

Figure 1: Macroscopic appearances of agglutination in round-bottom tubes or hollow tiles. Agglutination is shown by various degrees of 'graininess'; in the absence of agglutination, the sedimented cells appear as a smooth round button, as on the extreme right.

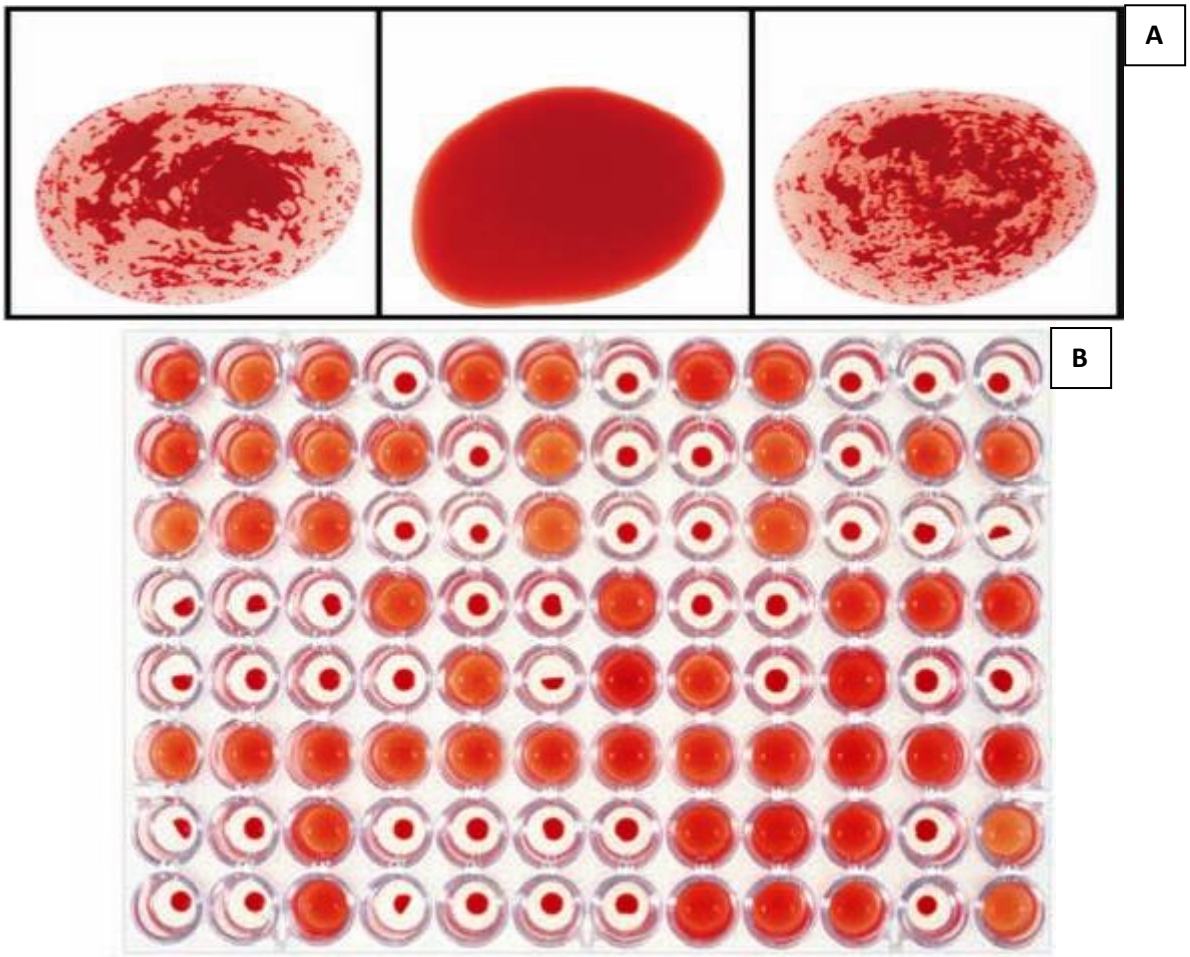

Figure 2: (A) The ABO grouping in a group A patient. The red cells suspended in saline agglutinate in the presence of anti - A or anti - A + B (serum from a group O patient). (B) Routine grouping in a 96 - well microplate Positive reactions show as sharp agglutinates; in negative reactions the cells are dispersed. Rows 1 – 3 patient cells against antisera rows 4 – 6, patient sera against known cells; rows 7 – 8, anti - D against patient cells.

RESULTS

Five hundred of blood donors attending the Central Blood bank in Wad Medani teaching hospital. Moreover the frequency percentages of the ABO blood group were O (51.%), A (30%), B (14%) and AB (5%) and the majority were Rh D positive (97.4%) and (2.6%) were negative. 97.3% of group A were Rh D positive and 2.7% were Rh D negative, 97.14% of group B were Rh D positive and 2.86% were Rh D negative, 97.3% of group O were Rh D positive and 2.7% were Rh D negative, 100% of group AB were Rh D positive and 0% were Rh D negative.

Table 1: Distribution of ABO blood group among blood donors

Blood group	Frequency	Percent
A	150	30
B	70	14
AB	25	5
O	255	51
Total	500	100.0

Table 2: Distribution of Rh D among blood donors with different ABO blood group types

Blood group	Frequency	Rh D	Percent
A	150	146	97.3
B	70	68	97.14
AB	25	25	100
O	255	248	97.3
Total	500	487	97.4

Table 3: Distribution of Rh D negative among blood donors with different ABO blood group types

Blood group	Frequency	Rh D negative	Percent
A	150	4	2.7
B	70	2	2.86
AB	25	0	0
O	255	7	97.3
Total	500	13	2.6

Table 4: Distribution of Rh D among blood donors

Blood group	Frequency	Percent
Rh D positive	487	97.4
Rh D negative	13	2.6
Total	500	100

DISCUSSION

Discovery of the ABO system by Landsteiner marked the beginning of safe blood transfusion. The ABO antigens, although most important in relation to transfusion, are also expressed on most endothelial and epithelial membranes and are important histocompatibility antigens.[3] Transplantation of ABO-incompatible solid organs increases the potential for hyperacute graft rejection, although ABO-incompatible renal transplantation can be successfully carried out with plasmapheresis in addition to immunosuppression of the recipient.[4] Major ABO-incompatible stem cell transplants (e.g. group A stem cells into a group O recipient) will provoke haemolysis, unless the donation is depleted of red cells. There are four main blood groups: A, B, AB and O. In the British Caucasian population, the frequency of group A is 42%, B 9%, AB 3% and O 46%, but there is racial variation in these frequencies. ABO antibodies are mostly immunoglobulin M, can be able to activate complement, which in conjugation with the high density of ABO antigens sites on RBCs, is responsible for severe, life threatening acute haemolytic transfusion reaction in case of ABO incompatible transfusion.

Laboratory analysis conducted in this study revealed that the percentage of ABO blood phenotypes in the total samples were as follows: O (51%), A (30%), B (14%), and AB (5%), this result are similar to some extend with world ABO distribution. The present study is the first comprehensive study that documents the frequencies of ABO, subgroup ABO and Rh (D) blood groups among Sudanese blood donors in Wad Medani.

CONCLUSIONS

1. This study shows that the most frequent ABO blood group is O followed by A, B and AB.
2. The majority of study population were Rh D positive.
3. Other study including other antigenic blood groups is highly recommended with increase the sample size.

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