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Occurrence of ABO and Rh D blood groups in Sudanese population attending blood bank, Gezira state, Wad Madani, Sudan, 2021

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Abstract

Objective: To detect the frequency of ABO and Rh D blood groups among Sudanese blood donors attending the Gezira center for Trauma and Orthopedic Blood Bank in Wad Madani.

Material and Methods: One thousand 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.

Keywords: ABO blood groups; Rhesus D; Blood Donors; Blood bank

1. Introduction

Blood is essential for transporting oxygen, nutrients, wastes and hormones in the body. The ABO blood group system is the most clinically important blood group system, which was discovered by Karl Landsteiner in 1900[1] and awarded the Nobel Prize in 1930. The fourth type of blood group was also discovered by Alfred Von Decastello and Adriano Sturli and named blood group AB, in 1902.[2] The rhesus (Rh) blood group system is the second most significant blood group system. Depending on the presence of the D antigen on the red cell, the blood group system has two phenotypes; RhD positive and RhD negative. The ABO and RhD genes are found at chromosomes 9 and 1, respectively. Proteins and carbohydrates, bided to lipids or protein, are the blood group determining red blood cells antigens.[3]

Even there are about 100 blood group systems with 500 antigens, the ABO blood group systems are the most clinically important blood group systems in blood transfusion and organ transplantation services.[4,5] A and B antigens are highly antigenic and have naturally occurring antibodies in human plasma which are missing the corresponding antigen and can cause hemolysis in vivo. The Rh blood group system is the second most clinically significant blood group with regard to transfusion. In addition to this, they are also important in the context of genetic studies, identifying of medico-legal issues and tracing family history.[5]

The presence of ABO antigens in red blood cells is depending on glycosyltransferases, which add sugars to the antecedent substance. However, a specific sets of epitopes and the RhD protein make up the D antigen.[6]

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The ABO blood groups are determined by using anti-sera (anti-A, anti-B and anti-AB) to detect the presence of A and B antigens. Additionally, known red cells antigens can be used to diagnose anti-A and anti-B in the plasma and called backward grouping. ABO and Rh phenotypes, allele and gene frequency differ broadly according to races and geographical borders.[3,7–9]

The frequency and distribution of ABO and Rh blood groups differ by ethnicity even in the same region. In Sudan, the frequencies of ABO and Rh blood groups have been investigated in some areas of the country, including Wad Madani in 2010

Detection of the Rh system is imperative to avoid the erythroblastosis fetalis; which frequently occurred when an RhD negative mother carries an RhD positive fetus and during the second birth causes the death of the fetus.

Knowing the frequency of ABO and RhD blood groups in a particular population is crucial for efficient management of blood bank inventor, even for local transfusion services.[9] Thus, the main objective of the current study was to determine the frequency and distribution of ABO and Rh blood groups among blood donors at the Wad Madani blood bank – Gezira state -Sudan.

1.1. 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 anti-gens(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.

1.2. 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.

1.3. 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 37 ? 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.

1.4. 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 Caucasianpopulation,about80%aresecretors(genotype SeSe or Sese) and20%arenon-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.

1.5. 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.

1.6. ABO Antibodies

1.6.1. 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 37 ? 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. [10]

They are predominantly of IgG class and are usually produced by group O and some times 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. [10]

1.6.2. 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 37 ? 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 37 ? C so that Oh Bombay phenotype blood would be required for transfusion. [10]

1.7. Lewis System

1.7.1. Lewis Antigens and Encoding Genes

The Lewis antigens (Le a and Le b) are located on soluble glycosphingolipids found in saliva and plasma and are secondarily absorbed into the red cell membranes from the plasma. The Le gene at the FUT3 (LE) locus is located on

chromosome19 and codes for a fucosyltransferase, which acts on an adjacent sugar molecule to that acted on by the Se gene. Where Se and Le are present, the Le b antigen is produced; where Le but not Se is present, Le a is produced; and where Le is not present, neither Le a nor Le b is produced.

After transfusion of red cells, donor red cells convert to the Lewis type of the recipient owing to the continuous exchange of glycosphingolipids between the plasma and red cell membrane. Neonates have the phenotype Le(a?b?) because low levels of the fucosyltransferase are produced in the first 2 months of life. [10]

1.7.2. Lewis Antibodies

Lewis antibodies are naturally occurring and are usually IgM and complement binding. In vitro, their reactivity is enhanced with the use of enzyme-treated red cells, when lysis may occur. However, only rare examples of anti-Le a that are strictly reactive at 37 ? C have given rise to haemolytic transfusion reactions and there is no good evidence that anti-Le b has ever caused a haemolytic episode.

Explanations for the relative lack of clinical significance include their thermal range, neutralization by Lewis antigens in the plasma of transfused blood and the gradual elution of Lewis antigens from the donor red cells.

Consequently, it is acceptable to provide red cells for transfusion that have not been typed as negative for the relevant Lewis antigen but are compatible with the recipient plasma when the compatibility test is performed strictly at 37 ? C.

Lewis antibodies have not been implicated in haemolytic disease of the fetus or newborn. The role of Lewis in influencing the outcome of renal transplants is unclear. [10]

1.8. The P System and Globoside Collection

1.8.1. Antigens

The P 1 antigen of the P system and the P and P k antigens of the globoside collection are related. Little is known of the genes involved or their products, but all are derived from the precursor, lactosyl ceramide dihexoside. Carbohydrate products related to the P system are widely distributed in nature. Expression of P 1 varies considerably between individuals. One in 100 000 individuals is p (negative for P) and is resistant to parvovirus B19 infection. [10]

1.8.2. Antibodies

Anti-P 1 is a common naturally occurring antibody of no clinical significance and red cells for transfusion can be provided that are crossmatch compatible at 37 ? C. Allo-anti-P is also a naturally occurring antibody found in individuals with the rare P k phenotype.

Auto-anti-P is the specificity attributed to the Donath–Landsteiner antibody; it is a potent biphasic haemolysin, responsible for paroxysmal cold haemoglobinuria.

Anti-PP 1 P k is a naturally occurring high-titre IgM or IgG antibody and it is found only in individuals with the rare p phenotype. It is reactive at 37 ? C and is capable of causing intravascular haemolysis and HDN. It is also associated with spontaneous miscarriage in early pregnancy. [10]

1.9. 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. [11]

1.10. 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. [11]

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. [15]

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. [15]

1.10.1. 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 37 ? 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. [11]

Primary immunization following a transfusion of D positive cells becomes apparent within2–5months,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. [11]

1.11. Kell and Kx Systems

1.11.1. Antigens and Encoding Genes

A total of 34 antigens have been identified (K1–K34), but three very closely linked sets of alleles are clinically important: K (KEL1) and k (KEL2); Kp a (KEL3), Kp b (KEL4) and Kp c (KEL21); and Js a (KEL6) and Js b (KEL7). These antigens are encoded by alleles at the KEL locus on chromosome 7, but their production also depends on genes at the KX locus on the X chromosome. The K antigen is present in 9% of the English population. The Kp b antigen has a high frequency in Caucasians; the Js b antigen is universal in Caucasians and almost universal in black races. The Kell protein is a single-pass glycoprotein and is believed to be complexed by a disulphide bridge to the Kx protein, which is multipass with 10 putative transmembrane domains. It has considerable sequence homology to other neutral endopeptidases. In the McLeod phenotype, red cells lack Kx and there is a marked decrease in all Kell antigens, an acanthocytic morphology and compensated haemolysis. The McLeod syndrome is X-linked with slow progression to cardiomyopathy, skeletal muscle wasting and neurological defects. [5]

1.11.2. Kell Antibodies

Immune anti-K is the most common antibody found out- side the ABO and Rh systems. It is commonly IgG1 and occasionally complement binding. Other immune antibodies directed against Kell antigens are less common. The presence of some of these antibodies, such as anti-k, anti- Kp b and anti-Js b, may cause extensive difficulties in the selection of antigen-negative units for transfusion. [5]

1.12. Duffy System

1.12.1. Duffy antigens and encoding genes

The Duffy (Fy) locus is on chromosome 1 and encodes a multipass protein with seven or nine putative transmembrane domains.

The locus has the following alleles: Fy a, Fy b, which code for the co-dominant Fy a and Fy b antigens, respectively; Fy x, which is responsible for a weak Fy b antigen; and Fy, which is responsible when homozygous for the Fy(a?b?) phenotype in black races.

This Fy gene is identical to the Fy b gene in its structural region but has a mutation in the promoter region, resulting in the lack of production of red cell Duffy glycoprotein.

The Fy glycoprotein (also known as Duffy antigen receptor for chemokines, DARC) is a receptor for the CC and CXC classes of proinflammatory chemokines and is expressed on vascular endothelial cells and Purkinje cells in the cerebellum, but its precise role as a potential scavenger of excess chemokines is unknown. The Fy glycoprotein is also a receptor for Plasmodium vivax. [5]

1.12.2. Duffy antibodies

Anti-Fy a is much more common than anti-Fy b and all other Duffy antibodies are rare apart from anti-Fy 3 (to both Fy a and Fy b), which occurs in some African/Afro Caribbean patients, in whom Fy(a?b?) antigen status is common. They are predominantly IgG1 and are sometimes complement binding.

1.13. Kidd (JK) System

1.13.1. Kidd antigens and encoding genes

Genes at the HUT 11(JK) locus on chromosome 18 encode for a multipass protein, which carries both the Kidd antigens and the human erythroid urea transporter. The codominant alleles, Jk a and Jk b, represent a polymorphism on HUT 11, which differs by a single amino acid substitution at position 280 (Asp/Asn). The Jk(a?b?) phenotype is very rare and is caused by homozygous inheritance of the silent allele, Jk, at the JK locus or by inheritance of the dominant inhibitor gene In (Jk)unlinked to the JKlocus. These Jk(a?b?) cells are resistant to lysis by solutions of urea and have a selective defect in urea transport. [5]

1.13.2. Kidd antibodies

Anti-Jk a is more common than anti-Jk b ; both are usually IgG. Kidd antibodies are usually complement binding, which is thought to be because most of them contain an

IgG3 fraction. Anti-Jk3 is produced by individuals of the rare Jk(a?b?) phenotype.

Kidd antibodies can be difficult to detect because they often show dosage (may only react with cells showing homozygous expressions of Jk a or Jk b),they fall to undetectable levels in plasma and they are often present in mixtures of alloantibodies.

A previous history of antibodies is therefore important, to avoid a post-transfusion haemolytic reaction, due to an anamnestic response by an antibody that was below the level of detection before transfusion. [5]

1.14. MNSs System

1.14.1. MNSs antigens and encoding genes

GYPA and GYPB are closely linked genes on chromosome 4 and encode glycophorin A (GPA) and glycophorin B (GPB), respectively. Both GPA and GPB are single-pass membrane sialoglycoproteins. M and N are alleles of GYPA (encoding the M and N antigens on GPA) and S and s are alleles of GPYB (encoding the S and s antigens on GPB). Many rare variants have been described owing to gene deletions, mutations and segmental exchanges. The U antigen is found on the red cells of Caucasians and 99% of black races. U-negative individuals are, with rare exceptions, S?s? and lack GPB or have an altered form of GPB. [5]

1.14.2. MNSs antibodies

Anti-M is a relatively common antibody that may be IgM or IgG. Rare examples are reactive at 37 ? C when they can give rise to haemolytic transfusion reactions. Anti-M very rarely gives rise to HDN. Anti-N is uncommon and of no clinical significance. Anti-S and anti-s are usually IgG; both rarely have been implicated in haemolytic transfusion reactions and HDN. Anti-U is a rare immune antibody, usually containing an IgG1 component. It has been known to cause fatal haemolytic transfusion reactions and occasional severe HDN. [5]

1.15. Other Blood Group Systems

1.15.1. Lutheran system

The antigens in the Lutheran system are not well-developed at birth and as a consequence there are no documented cases of clinically significant haemolytic disease owing to. [5]

1.15.2. Lutheran antibodies

Anti-Lu a is uncommon and rarely of clinical significance. Anti-Lu b has caused extravascular haemolysis. Yt (Cartwright) system. The antigens Yt a and Yt b are found on GPI-linked acetyl-cholinesterase. Some examples of anti-Yt a have caused accelerated red cell destruction. [5]

1.15.3. Colton system

The antigens in the Colton system, Co a and Co b, are carried on the water-transport protein, channel-forming integral protein (CHIP-1). Anti-Co a and the rarer anti-Co b are both sometimes clinically significant. [5]

1.15.4. Dombrock system

The antigens in the Dombrock system include Do a and Do b and also include the high-incidence antigens Gy a, Hy and Jo a . Antibodies of this system are usually weak, but all should be considered as potentially significant. [5]

1.16. Clinical Significance of Red Cell

1.16.1. Alloantibodies

The majority of haemolytic transfusion reactions, however, are the result of ABO incompatibility. [16] Mollisonetal. [17] analysed the significance of blood group antigens other than those of the ABO system and D by looking at the prevalence of transfusion-induced red cell alloantibodies, excluding anti-D, -CD and Rh antibodies, mainly anti-c or anti-E, accounted for 53% of the total and anti-K and anti-Fy a accounted for a further 38%, leaving only about 9% for all other specificities.

A similar distribution of the different red cell antibodies was found in a smaller group of patients who experience immediate haemolytic transfusion reactions (HTR). However, the figures for delayed HTR showed a striking increase in the relative frequency of Jk antibodies, which reflects the outlined characteristics of Jk antibodies. Haemolytic disease of the fetus and newborn has not been associated with antibodies directed against Lewis antigens and only very mild disease is produced by anti-Lu a and anti-Lu b. With these exceptions, all other IgG antibodies directed against antigens in the systems mentioned should be considered capable of causing haemolysis in this setting. However, it should be noted that the antibodies listed are usually wholly or predominantly IgG and would be detectable in routine pretransfusion testing using the indirect anti-globulin test (IAT). It is difficult to find suitable blood for transfusion to a patient whose plasma contains an antibody, such as anti-Vel, which has a specificity for a high-frequency antigen and which can cause severe haemolytic transfusion reactions. In addition to using blood from a frozen blood bank and calling up rare phenotype donors, autologous blood could be considered for planned elective procedures and if necessary, the compatibility of red cells from close relatives (particularly siblings) can be investigated. Antibodies such as anti-Kn a are commonly found and not clinically important, but their presence may cause delay in the provision of blood until their specificity has been determined. [17]

1.17. 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.

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

Individuals with fever, high blood pressure, very high or very low heart rate (with the exception of highly conditioned athletes and those on beta blocker medication), or an irregular heart beats are temporarily not permitted to donate blood. **[18]**

Seizures: People with a history of seizures can donate blood, provided they have had no seizures within a certain period of time (usually 1 to 6 months).

Recent surgery: People who have undergone recent surgery are permitted to donate blood when healing is complete and they have resumed full activity. However, if a transfusion was given at the time of surgery, donation is not allowed for one year.

1.18. Pregnancy

Women who are pregnant usually not accepted as blood donors during pregnancy and for six weeks after the pregnancy ends. **[18]**

People who have received a blood transfusion are not permitted to donate blood for 12 months after the date of transfusion.

People who take aspirin or aspirin-containing medications within the previous 48 hours are allowed to donate whole blood but are not allowed to donate platelets by aphaeresis.

People who take warfarin (Coumadin) are generally not allowed to donate blood for approximately 7 days after the last dose. The reason for this is that this blood does not contain sufficient quantities of clotting factors. [18] Individuals with high risk history (permanent) of HIV confirmed positive test for hepatitis B surface antigen(HBs Ag), positive test for hepatitis B core antibody, confirmed positive test for hepatitis C antibody (HC Ab), confirmed positive test for Human T-cell lymphotropic virus (HTLV 1-2). Malignant solid tumors, haematological malignancies, chronic cardiopulmonary, liver and renal diseases, serious abnormal bleeding disorders are not allowed to donate blood.

1.18.1. Age requirement

The minimum age for blood donation is 17 years and the maximum is 60 years. Persons younger than 17 years donate with written permission from their legal guardian. [18]

Weight requirement: Individuals weighing less than 50 kg (110 pounds) are usually not permitted to donate blood. The less a donor weighs, the greater the likelihood of having a reaction, such as dizziness and fainting following donation. [18]

1.18.2. 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. [19]

1.18.3. 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 clinical 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 initial testing must be examined by the antihumanglobulin (AHG) test, to detected the D^U variant (weak D) all the ABO and Rh (D) negative units must be confirmed by the hospital transfusion service prior to transfusion. [19]

1.18.4. 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. [20]

1.19. Cross matching

1.19.1. Indirect Antiglobulin Crossmatch

The arguments for retaining an indirect antiglobulin crossmatch are based on the failure to identify antibodies against low-frequency antigens in the antibody screen. Moreover, the usual outcome of transfusion if an antibody is present but undetected by the antibody screen is limited to shortened red cell survival. An indirect antiglobulin crossmatch should always be performed if the patient has red cell antibodies of likely clinical significance, even if currently undetectable. The reasons for this are as follows:

- It acts as a double check that the donation has been correctly phenotyped and labelled as negative for the corresponding antigen(s).
- It ensures serological compatibility even if the identification of the antibodies is incorrect or incomplete.

- It allows detection of antibodies to low-frequency antigens not present on the screening cells, which may be more likely to be present in a patient who is clearly a "responder" and which may be masked by other alloantibodies Methods for indirect antiglobulin crossmatching are the same as those used for antibody screening. Crossmatching may be less effective than antibody screening at detecting incompatibility as a result of IgG antibodies. This is partly because the cells may only show heterozygous expression of an antigen to which the patient has an antibody, potentially leading to weaker or negative reactions, and also because the cell suspensions and other techniques (e.g., cutting pigtails from donations, labelling tubes, washing cells) are less likely to be standardized and present more opportunity for transposition errors than the screening processes.

1.19.2. Immediate Spin Crossmatch

The sole purpose of this technique is to detect ABO incompatibility. It can be used in order to issue blood for transfusion when the patient has a full blood group and negative antibody screen. It may be used to convert a group and save/screen when blood is required and is often used in addition to an antiglobulin crossmatch.

The immediate spin crossmatch will ensure that the correct units have been selected and that the correct ABO group has been assigned to the unit of donor red cells. Clearly it is not a suitable test to use for detection of ABO incompatibility if the reverse blood group reveals a very weak anti-A or anti-B, or if the patient falls into one of the categories described in the previous section. There is evidence of poor standardization of this technique, but its sensitivity can be optimized by selecting the appropriate cell suspension, incubation time, and serum : cell ratio. The following tube method is recommended:

1.19.3. False-Negative Results

Incompatibilities between A_2B donor cells and group B patient sera are not consistently detected with this technique. Of more concern is the potential failure of agglutination with potent ABO antibodies on account of rapid complement fixation with bound C1 interfering with agglutination—if using serum, red blood cells must be suspended in saline containing EDTA.

1.19.4. False-Positive Results

Cold reacting antibodies other than anti-A and anti-B may cause agglutination in an immediate spin crossmatch. This has the potential to cause delays to transfusion while further procedures are used to rule out ABO incompatibility.

1.19.5. Electronic Issue

In many countries electronic issue is a commonly used alternative to the immediate spin crossmatch for issuing blood for transfusion when the patient has had a full blood group and no history of clinically significant antibodies. ABO and D compatible units are selected and issued through a computer system, which contains logic rules to prevent the issue of ABO and D incompatible blood. [21]

1.19.6. Screening for infections

Donated blood is tested by many methods, but the core tests recommended by the World Health Organization are:

- Hepatitis B Surface Antigen.
- Antibody to Hepatitis C.
- Antibody to HIV, usually subtypes 1 and 2.

Serologic test for Syphilis. [21]

2. Methods

2.1. Sample collection

A total of 1000 apparently healthy adult male donors were screened for ABO blood groups. This analysis was conducted at the Wad Madani, Gezira center for Trauma and Orthopedic Blood Bank, department of pathology (Medical laboratory) from June 2021 to December 2021.

2.2. 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.

2.3. Interpretation of result

Results of agglutination were recorded immediately for ABO blood groups and after 2 minutes in Rh (D). Mixed well over an area of 2.5cm. 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.

2.4. 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.

2.5. Ethical clearance

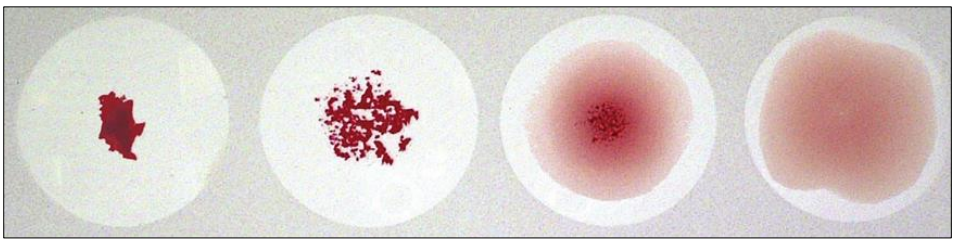

Figure 1 Macroscopic appearances of agglutination in round-bottom tubes or hollow tiles: Agglutination is shown by various degrees of 'graininess'; in the absence of agglutination, the sedimented cells appear as a smooth round button, as on the extreme right

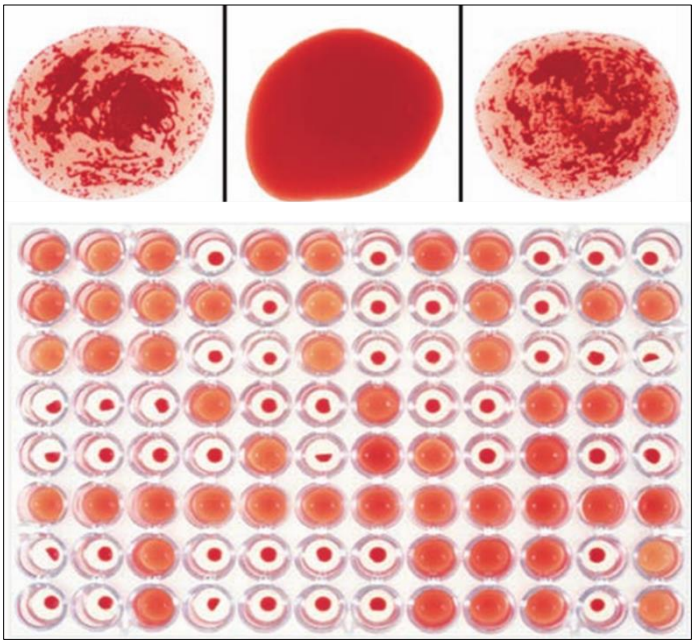

Figure 2 (A) The ABO grouping in a group A patient. The red cells suspended in saline agglutinate in the presence of anti - A or anti - A + B (serum from a group O patient) (B) Routine grouping in a 96 - well microplate Positive reactions show as sharp agglutinates; in negative reactions the cells are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

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

3. Results

One thousands of blood donors attending the Central Blood bank in Wad Madani teaching hospital. Moreover the frequency percentages of the ABO blood group were O (50.2%), A (31.2%), B (13.4%) and AB (5.2%) 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, 96.9% of group O were Rh D positive and 3.1% were Rh D negative.

Table 1 Distribution of ABO blood group among blood donors

Blood group	Frequency	Percent
A	312	31.2
B	134	13.4
AB	52	5.2
O	502	50.2
Total	1000	100.0

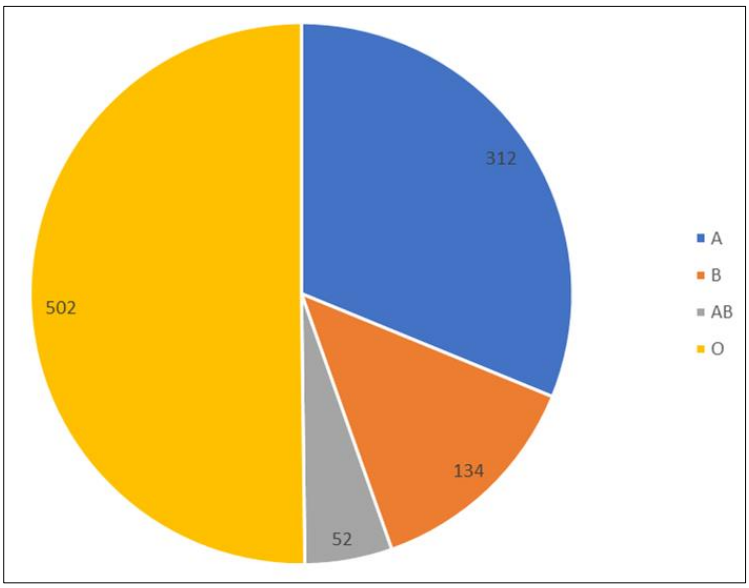

Figure 3 Distribution of ABO blood group among blood donors

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

Blood group	Frequency	Rh D	Percent
A	312	300	96.2
B	134	128	95.5
AB	52	50	96.2
O	502	491	97.8
Total	1000	969	96.9

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

Blood group	Frequency	Rh D negative	Percent
A	312	12	3.8
B	134	6	4.5
AB	52	2	3.8
O	502	11	2.2
Total	1000	31	3.1

Table 4 Distribution of Rh D among blood donors

Blood group	Frequency	Percent
Rh D positive	969	96.9
Rh D negative	31	3.1
Total	1000	100

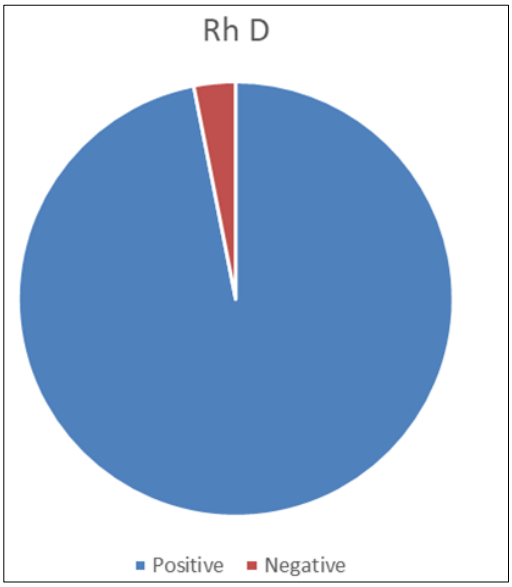

Figure 4 Distribution of Rh D among blood donors

4. 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 (50.2%), A (31.2%), B (13.4%), and AB (5.2%), this result are similar to some extend with world ABO distribution

5. Conclusion

This study showed that the most frequent ABO blood group is O followed by A, B and AB and the majority of study population were Rh D positive.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors do have not any conflicts of interest in this case report and any financial resources.

Statement of ethical approval

Ethical approval was obtained from the University of Gezira ethical committee and blood bank authority.

Statement of informed consent

Informed consent and verbal permission were obtained from the donor before the submission of this article.

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