

BLOOD GROUPS SYSTEMS

Mrs.E.Veena

Department of Biomedical Engineering, SNS College of Technology, Coimbatore

Mr.T.Arun Kumar and Dr.D.Shalini

Department of Chemistry, SNS College of Engineering, Coimbatore

Abstract

International Society of insertion has recently recognized thirty three blood type systems. Except for Aborigine and rhesus monkey system, several different forms of antigens are detected on the red cell membranes. Blood grouping and cross-matching is one in every of the few necessary tests that the anesthesiologists orders throughout per operative amount. Hence, a correct understanding of the blood type system, their clinical significance, writing and cross-matching tests, and current perspective are of predominate importance to forestall transfusion-related complications. Still, the information on blood type system is critical to approach blood group-linked diseases that are still at the stage of analysis. This review addresses of these aspects of the blood teams system.

Keywords: *Aborigine blood teams, protein writing, blood type system, rhesus monkey blood type, screening*

Introduction

The term “blood cluster” refers to the complete blood type system comprising red somatic cell (RBC) antigens whose specificity is controlled by a series of genes which might be gene or connected terribly closely on a similar body. “Blood type” refers to a selected pattern of reaction to testing antisera among a given system. Over a amount of your time, our understanding on blood teams has evolved to comprehend not solely transfusion-related issues however conjointly specific malady association with erythrocyte surface antigens. pathologist has been attributable for the invention of Australian Aborigine blood type system in 1900.[1] His intensive analysis on medical science supported easy however robust scientific reasoning semiconductor diode to identification of major blood teams like O, A, and B sorts, compatibility testing, and resulting transfusion practices. He was awarded Noble Prize in 1930 for this discovery. His promulgation lists Associate in nursing huge contribution of quite 346 publications. Later, Jan Jansky delineates classification of human blood teams of 4 sorts.

Blood Groups

At present, thirty three blood type systems representing over three hundred antigens are listed by the International Society of intromission.[2,3] Most of them are cloned and sequenced. The genes of that blood type systems ar chromosome, except XG and XK that are X-borne, and MIC2 that is gift on each X and Y chromosomes. The antigens will be integral proteins wherever polymorphisms consist the variation of aminoalkanoic acid sequence (e.g., rhesus monkey [Rh], Kell), glycoproteins or glycolipids (e.g., ABO). A number of the necessary team's are mentioned here [Table 1].

Table 1

Name	Symbol	Number of antigens	Gene name	Chromosome
ABO	ABO	4	ABO	9
MNS	MNS	43	GYPA, GYPB, GYPE	4
P	P1	1	P1	22
Rhesus	Rh	49	RhD, RhCE	1
Lutheran	LU	20	LU	19
Kell	KEL	25	KEL	7
Lewis	LE	6	FUT3	19
Duffy	FY	6	FY	1
Kidd	Jk	3	SLC14A1	18

Table 1 Blood group systems**ABO system**

Among the 33 systems, ABO remains the foremost necessary in transfusion and transplantation since a person higher than the age of 6 months possess clinically important anti-A and/or anti-B antibodies in their humor. People A contains protein against people B in humor and vice-versa, whereas people O contains no A/B matter however each their antibodies in humor

H-antigen

H-antigen is that the precursor to the ABO blood type antigens. it's gift altogether RBCs no matter the ABO group. Persons with the rare Bombay makeup are homozygous for the H cistron (HH), don't categorical H-antigen on their RBCs. As H-antigen acts as precursor, its absence suggests that the absence of substance A and B. However, the people turn out iso antibodies to H-antigen further on antigens A and B.

Rhesus System

Rhesus-system is that the second most vital blood type system when ABO.[4] presently, the Rh-system consists of 50 outlined blood type antigens out of that solely 5 are necessary. Erythrocyte surface of a person might or might not have a antigen or immunogenic D-antigen. Consequently, the standing is indicated as either Rh-positive (D-antigen present) or Rh-negative (D-antigen absent). In distinction to the ABO group, anti-Rh antibodies are, normally, not gift within the blood of people with D-negative RBCs, unless the cardiovascular system of those people has been exposed to D-positive RBCs. These immune antibodies are immune gamma globulin (IgG) in nature and thence, will cross the placenta. Bar is given against Rh immunisation mistreatment anti-D human gamma globulin for pregnant Rh-negative mothers UN agency has born to Rh-positive kid.

MNS Antigen System

MNS matter system, 1st delineates by diagnostician and Levine in 1927 relies on 2 genes: Glycophorin A and Glycophorin B. The people are in restraint of associate degree chromosome locus on body four and conjointly in restraint of a combine of co-dominant allele's luminous

flux unit and LN. Anti-M and anti-N antibodies square measure typically Ig sorts and barely, related to transfusion reactions.

Lutheran System

Lutheran system comprised of 4 pairs of allelomorphous antigens representing single organic compound substitution within the Lutheran conjugated protein at body nineteen. Antibodies against this people square measure rare and usually not thought-about clinically important.

Kell System

These corpuscle matters square measure the third most potent immunogenic antigen once ethnos and Rh system, associate degree square measure outlined by an immune protein, anti-K. it had been 1st noticed within the body fluid of Mrs. Kellacher. She reacted to the erythrocytes of her baby leading to haemolytic reactions. Since then 25Kell antigens are discovered. Anti-K protein causes severe haemolytic illness of the fetus and newborn (HDFN) and hemolytic transfusion reactions (HTR).

Duffy system

Duffy-antigen was 1st isolated in a very patient known as Duffy UN agency had bleeder's disease. It conjointly referred to as Fy conjugated protein and is gift within the surface of RBCs. it's a nonspecific receptor for many chemokines and acts as a receptor for human protozoal infection parasite, *Plasmodium vivax*. Antigens FyA and FyB on the Duffy conjugated protein may result in four doable phenotypes, particularly Fy(A⁺B⁻), Fy(A⁺B⁺), Fy(A⁻B⁺), and Fy(A⁻B⁻). The antibodies square measure immunoglobulin G subtypes and might cause HTR.

Kidd System

Kidd substance (known as Jk antigen) could be a conjugated protein, gift on the membrane of RBCs and acts as a organic compound transporter in RBCs and nephritic epithelium cells. William Kidd antibodies square measure rare however will cause severe transfusion reactions. These Antigens square measure outlined by reactions to a protein selected as anti-Jka, discovered within the humor of Mrs. William Kidd WHO delivered a baby with HDFN. Jka was the primary substance to be discovered by William Kidd blood type system, later on, 2 alternative antigens Jkb and Jk3 were found.

Agarwal et al. [5] administered a study on machine-controlled analysis of blood teams in north Indian donor population and determined that the common blood teams so as of frequency were B, O, A, and AB; 94.4% being Rh-positive. In minor blood teams, the foremost usually showing phenotypes were autoimmune disorder (A⁻B⁻) for Lewis, Fy(A⁺B⁺) for Duffy, Jk(A⁺B⁺) for William Kidd, and M⁺N⁺ for MNS system.

Importance of Blood Groups

Structural lesions of red blood Cell of the 33 blood type system antigens, 5 square measure outlined by their super molecule structures (ABO, H, P1Pk, I, GLOB); 2 square measure

obtained from the plasma (LE, CHRG). The remaining twenty three square measure characterized by the super molecule sequence of the red blood cell membrane super molecule,[6,7] 5 major proteins (DI, Rh, RhAG, MNS, GE, and CO) among them square measure expressed at higher levels and performance as membrane transporters, whereas the practical importance of remainder of seventeen antigens is unknown. The projected operate of alternative antigens square measure principally receptor/ligand communication, catalyst activity, and glycocalyx formation.[8] The null constitution of the system, however, shows no system abnormalities in comparison with mice apart from a dull neutrophile response on exposure to microorganism lipopolysaccharide.[9] equally, Knops blood type substance has been related to complement receptor 1[10] and Cromer system with decay acceleration issue.[11] nonetheless, the clinical operate of the null phenotypes of those blood teams still remains to be elucidated [Table2].

Blood group system	Protein products	Function	Functional lesion in RBC in null phenotypes	Clinical significance
DI	Band-3	Anion transport	Hereditary spherocytosis	Growth retardation Distal renal tubular acidosis
MNS	Glycophorin A Glycophorin B	Facilitates membrane assembly of band-3	No lesions	
Rh	D polypeptide CE polypeptide	Facilitates band 3/RhAG complex assembly	Hereditary stomatocytosis	Increased permeability to cations
RhAG	Rh associated glycoprotein	Neutral gas transport	Hereditary stomatocytosis	Increased permeability to cations
GE	Glycophorin C Glycophorin D	Maintains red cell shape through interaction with protein 4.1	Hereditary ellipsocytosis	
CO	Aquaporin-1	Water/CO ₂ transport	Impaired water transport	
LY	Duffy glycoprotein (DARC)	Chemokine receptor for proinflammatory cytokines	Absence of chemokine binding	
Kell	Kell glycoprotein	Zinc endopeptidase	No lesion	
Jk	Urea transporter	Urea transporter	Reduced urea transport	
Cromer	Decay accelerating factor (CD55)	C3 convertase inhibitor	No lesion	
IU	I antigen glycoprotein	Ligand for laminin 511/512	No lesion	
XK	Kx glycoprotein	Amino acid transport	Acanthocytosis	Medeoid phenotype (CNS abnormalities, muscle weakness, cardiomyopathy)

CO₂ = Carbon dioxide; CNS = Central nervous system; RBC = Red blood cell; Rh = Rhesus; CO = Colton; GE = Gerbich; DI = Diego

Table 2 Pathology Related to the Null Constitution of the Red Blood Cell Substance

Blood Teams and Illness Association

The Aussie blood teams have a profound influence on stop.[12] They exert major quantitative effects on plasma levels of Erik Adolf von Willebrand issue and Hemofil. Accumulated association of MI, apoplexy, and blood vessel occlusion is seen with blood teams A and AB[13] probably through practical Aussie glycol transferases modulation of occlusion. a better risk of cerebral thrombosis has been according in non-O teams.[14] important association of Aussie teams with the prevalence of toxemia of pregnancy has been according, wherever AB cluster was found to be related to AN accumulated risk of two.1-folds.[15] Preliminary studies urged AN association of ABO system with malignancies. A correlation statistics has been shown between blood type A with chronic hepatitis-B infection and duct gland cancer; [16] and blood type B with gonad cancer.[17] Protection against falciparum protozoal infection are often achieved with type O by reducing rosette formation.[18] blood type O will increase the severity of infection in eubacterium cholerae strains (O1 El Tor and O139).

Blood Requisition

After the choice to transfuse blood is taken subsequent step ought to be to order a requisition throughout that the subsequent steps ought to be remembered.

Blood Grouping and Cross-Matching

The most fatal of all transfusion-related reaction is Aussie incompatibility inflicting complement-mediated intravascular haemolysis. Hence, correct blood grouping and typewriting, and cross-checking with the blood form is of utmost importance. Aussie typewriting is administered by testing RBCs for the A and B antigens and also the humor for the A and B antibodies before transfusion. Subsequent step involves Rh typewriting with solely V-day of the population being Rh-negative.

Cross-Matching

Cross-matching involves combining of donor RBCs with the recipient humour to find fatal reactions.[19] it's 3 parts during which the primary phase (1-5 min) involves detection of Australian incompatibility and detection of protein against MN, P, and Lewis systems. The second part (30-45 min in albumen and 10-20 min in low ionic salt solution) involves incubation of initial part reactants at 37°C for detection of incomplete antibodies of Rh system. The third part consists of the addition of antiglobulin sera to the incubated second part reactants to find incomplete antibodies of Rh, Kidd, Kell and Duffy. Among the 3 phases, the primary 2 phase's ar a lot of necessary as they find those concerned in fatal HTR. the full time taken for all the 3 phases is in between 45and 60min.

Antibody Screening

Here, commercially ready RBCs with all the antigens, that direct production of antibodies inflicting lysis reactions, ar mixed with the recipient's humour to find the presence of these terribly antibodies. It's conjointly dispensed with the donor's humour.

Changing Practices in Blood Grouping

There are controversies relating to the simplest methodology for procural of blood throughout elective and emergency situations: (a) It are often done by habitually requesting grouping and cross-matching in elective surgical patients. Several scientific articles controversial the connection of operative arrangement of blood in surgeries wherever blood loss isn't anticipated to be important. [20, 21] (b) Blood could also be ordered while not full set of investigations.[19] ABO-Rh typewriting alone ends up in a ninety nine.8% probability of a compatible transfusion. Protein screening will increase this margin of error up to99.94%, and additional an extra} cross-match further will increase the compatibility to ninety nine.95%. In absence of cross-matching, there's a break of missing the antigens on donor cells, however in clinical apply, they're of less importance. Hence, "screening and typing" alone ought to be dispensed. alternative strategies embody "type and partial cross-match," which incorporates the immediate part of cross-match; "type and open match," for those recipients United Nations agency haven't been transfused before, the prospect of detection of protein with every cross-match is 1:1000; "type O rh-negative open match," it's performed in emergency scenario once the time for these procedures is restricted. within the latter condition, type O rh-negative

packed RBCs, that is, the blood donor are often used as they're going to have a negligible quantity of lysis anti-A/anti-B antibodies against the recipient RBCs.

Current Trends and Future Areas of

Research Three main antigen-modulation methods are planned to forestall immune recognition of incompatible RBCs and to avoid hemolytic reactions as a result of allow immunization. The primary approach depends on catalyst conversion of specific people antigens, that is, manipulation of the ABO system. Goldstein and Lenny achieved an interesting milestone with the event of technology named "enzyme born-again cluster O-RBC (ECO-RBC) concept" wherever the B matter is replaced with O victimization galactosidase.[22] This treatment leaves fewer than 2000 substance sites per blood corpuscle while not touching membrane deformability, gas exchange, or expression of the Red Hand Defenders, C and E, MNS, Lewis, Kell, Lutheran, Duffy, and Kidd people systems as their antigenicity don't rely on the terminal sucrose residues. In distinction with the B matter, catalyst conversion of A matter was troublesome as a result of existence of 2 Type-A people structures (A_2 and A_1).[23] 2 new enzymes, N-acetylgalactosaminidase and a-galactosidase are known for removal of antigens A and B, respectively; and tested for his or her ability to come up with ECO-RBCs from A_1 , A_2 , B, or AB donor units.[24] The catalyst conversion strategy has conjointly been planned to resolve Australian incompatibility problems within the field of organ transplantation.[25] The second approach is to mask antigens by treatment of blood corpuscles with synthetic resin glycol; conjointly called the stealing RBC thought. The third approach involves in vitro production of RBCs with a predefined substance profile from genetically manipulated stem cells. [26] Such cells can be used for the generation of "universal-donor" RBCs.

Summary

Currently, our information on blood teams goes on the far side the standard tests of agglutination and transfusion to the higher understanding of corpuscle antigens in light-weight of their association with multiple illnesss and also the scope of use of this data to modulate the disease processes. During this context, the role of adequate understanding of screening, typing, and cross-matching aside from awareness on evolving trends, for each practician, might not be overemphasized. Footnotes Source of Support: Nil, Conflict of Interest: None declared

References

1. Owen R. Karl Landsteiner and the first human marker locus. Genetics. 2000; 155:995–8. [PMC free article] [PubMed]
2. Lögdberg L, Reid ME, Lamont RE, Zelinski T. Human blood group genes 2004: Chromosomal locations and cloning strategies. Transfus Med Rev. 2005; 19:45–57. [PubMed]
3. Lögdberg L, Reid ME, Zelinski T. Human blood group genes 2010: Chromosomal locations and cloning strategies revisited. Transfus Med Rev. 2011; 25:36–46. [PubMed]

4. Westhoff CM. The Rh blood group system in review: A new face for the next decade. *Transfusion*. 2004; 44:1663–73. [PubMed]
5. Agarwal N, Thapliyal RM, Chatterjee K. Blood group phenotype frequencies in blood donors from a tertiary care hospital in north India. *Blood Res*. 2013;48:51–4. [PMC free article] [PubMed]
6. Anstee DJ. The functional importance of blood group-active molecules in human red blood cells. *Vox Sang*. 2011; 100:140–9. [PubMed]
7. Daniels G, Reid ME. Blood groups: The past 50 years. *Transfusion*. 2010; 50:281–9. [PubMed]
8. Denomme GA. The structure and function of the molecules that carry human red blood cell and platelet antigens. *Transfus Med Rev*. 2004;18:203–31. [PubMed]
9. Luo H, Chaudhuri A, Zbrzezna V, He Y, Pogo AO. Deletion of the murine Duffy gene (Dfy) reveals that the Duffy receptor is functionally redundant. *Mol Cell Biol*. 2000; 20:3097–101. [PMC free article] [PubMed]
10. Rao N, Ferguson DJ, Lee SF, Telen MJ. Identification of human erythrocyte blood group antigens on the C3b/C4b receptor. *J Immunol*. 1991;146:3502–7. [PubMed]
11. Telen MJ, Hall SE, Green AM, Moulds JJ, Rosse WF. Identification of human erythrocyte blood group antigens on decay-accelerating factor (DAF) and an erythrocyte phenotype negative for DAF. *J Exp Med*. 1988;167:1993–8. [PMC free article] [PubMed]
12. Zhang H, Mooney CJ, Reilly MP. ABO Blood Groups and Cardiovascular Diseases. *Int J Vasc Med* 2012. 2012;641917. [PMC free article] [PubMed]
13. Wiggins KL, Smith NL, Glazer NL, Rosendaal FR, Heckbert SR, Psaty BM, et al. ABO genotype and risk of thrombotic events and hemorrhagic stroke. *J Thromb Haemost*. 2009;7:263–9. [PMC free article] [PubMed]
14. Tufano A, Coppola A, Nardo A, Bonfanti C, Crestani S, Cerbone AM, et al. Non-O blood group as a risk factor for cerebral vein thrombosis. *Thromb Haemost*. 2013;110:197–9. [PubMed]
15. Hiltunen LM, Laivuori H, Rautanen A, Kaaja R, Kere J, Krusius T, et al. Blood group AB and factor V Leiden as risk factors for pre-eclampsia: A population-based nested case-control study. *Thromb Res*. 2009;124:167–73. [PubMed]
16. Wang DS, Chen DL, Ren C, Wang ZQ, Qiu MZ, Luo HY, et al. ABO blood group, hepatitis B viral infection and risk of pancreatic cancer. *Int J Cancer*. 2012;131:461–8. [PubMed]
17. Gates MA, Wolpin BM, Cramer DW, Hankinson SE, Tworoger SS. ABO blood group and incidence of epithelial ovarian cancer. *Int J Cancer*. 2011;128:482–6. [PMC free article] [PubMed]
18. Anstee DJ. The relationship between blood groups and disease. *Blood*. 2010;115:4635–43. [PubMed]
19. Miller RD. Transfusion therapy. In: Miller RD, Ericksson LI, Fleischer LA, Weiner-Kronish JP, Young LA, editors. *Miller's Anesthesia*. 7th ed. Philadelphia: Churchill Livingstone Elsevier; 2010. pp. 1739–66.

20. Ghirardo SF, Mohan I, Gomensoro A, Chorost MI. Routine preoperative typing and screening: A safeguard or a misuse of resources. *JSLs*. 2010;14:395–8. [PMC free article] [PubMed]
21. Onotai L, Lilly-Tariah OD. Adenoid and tonsil surgeries in children: How relevant is pre-operative blood grouping and cross-matching? *Afr J Paediatr Surg*. 2013;10:231–4. [PubMed]
22. Goldstein J, Siviglia G, Hurst R, Lenny L, Reich L. Group B erythrocytes enzymatically converted to group O survive normally in A, B, and O individuals. *Science*. 1982;215:168–70. [PubMed]
23. Goldstein J. Conversion of ABO blood groups. *Transfus Med Rev*. 1989;3:206–12. [PubMed]
24. Liu QP, Sulzenbacher G, Yuan H, Bennett EP, Pietz G, Saunders K, et al. Bacterial glycosidases for the production of universal red blood cells. *Nat Biotechnol*. 2007;25:454–64. [PubMed]
25. Kobayashi T, Liu D, Ogawa H, Miwa Y, Nagasaka T, Maruyama S, et al. Alternative strategy for overcoming ABO incompatibility. *Transplantation*. 2007;83:1284–6. [PubMed]
26. Hashemi-Najafabadi S, Vasheghani-Farahani E, Shojaosadati SA, Rasaei MJ, Armstrong JK, Moin M, et al. A method to optimize PEG-coating of red blood cells. *Bioconj Chem*. 2006;17:1288–93. [PubMed]