Study on Blood Group and their Forensic Aspects

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Abstract

Prior to the widespread adoption of DNA typing, the blood group system was an important component in forensic serological examination of blood and body fluids. Blood grouping is one of the most important aspects in the term of forensic investigation because the blood plays a very crucial role when it comes to individualization and identification. It is one of the most reliable source and one of the most common evidence which found on the crime scene. The condition in which the blood is present or encountered on the crime scene may vary. It helps in the identification of the person with respect to its height in the case of the spattering of the blood or blood spatter analysis. It also link suspects to the crime scene and also narrows down the investigation process.

Keywords: Blood group; MNS; Kell group; Bombay blood group; Golden blood group.

INTRODUCTION

Landsteiner discovered the ABO blood types in 1901. Landsteiner and Wiener discovered Rhesus blood types in 1940, after discovering them in 1939. More than 20 different blood group systems have been identified since 1901, although the ABO and Rhesus blood groups are still the most clinically important. They're also well defined genetic markers that are used in population genetics. The expression "blood group" alludes to the whole blood group containing red blood cells (RBC) antigens whose particularity is constrained by a progression of gene which can be allelic or connected intently on a similar chromosome. "Blood classification" alludes to a particular example of response to testing antisera inside a given group. Throughout some stretch of time, our comprehension on blood groups has advanced to envelop bonding related issues as well as explicit illness relationship with RBC surface antigens.[1] The presence or absence of an inherited antigenic component on the surface of red blood cells, which may be identified by certain antibodies, determines the blood group or blood type. The importance of blood group discovery can be seen in blood transfusions between people of different ethnic backgrounds, organ transplantation, and the advancement of legal medicine, genetic research, and anthropology. The presence or absence of A and B surface antigens divides the primary ABO blood group system into four blood types. A, B, O, and AB are the blood types. The prevalence of four major ABO blood types varies greatly around the globe.[1]

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The importance of the ABO blood group system stems from the fact that A and B are highly antigenic, and anti A and anti B antibodies naturally occur in the serum of people who lack the corresponding antigen, and these antibodies are capable of causing intravascular hemolysis in the event of an incompatible transfusion. Hirschfled, who assessed blood types in a significant number of soldiers, including Indians, during the First World War, initiated blood group investigations on this subcontinent in 1919, and discovered a high prevalence of blood group B. Despite the fact that records for endogamous population groups were not kept separately, the research indicated significant geographical and ethnic disparities in blood group frequency.[2]

Within a given system, "blood type" refers to a certain pattern of reaction to antisera testing. Our knowledge of blood groups has grown over time to include not only transfusion related issues, but also particular illness associations with RBC surface antigens. In 1900, Karl Landsteiner is credited with discovering the ABO blood group system. His considerable research on serology led to the identification of major blood groups such as O, A, and B types, compatibility testing, and subsequent transfusion techniques, all based on simple but sound scientific logic. In 1930, he was awarded the Noble Prize for this finding. In his obituary, he is credited with more than 346 publications. Jan Jansky later described the classification of four types of human blood groupings.[3,4]

WHAT MAKE BLOOD TYPE?

The following are the key components of blood:

- Red blood cells, which are responsible for transporting oxygen throughout the body.
- White blood cells, which play an important function in the immune system plasma, a yellowish liquid containing proteins and ions.
- Platelets, which are responsible for clotting.

The blood group is determined by the antigens found on the red blood cell surface.

Antigens are a type of molecule. Proteins or carbohydrates can be found in them. Antigen kinds and characteristics can differ between people due to minor genetic variances.[5]

Antigens in blood serve a variety of purposes, including

Other molecules are transported into and out of the cell, sustaining the red blood cell structure, discovering unwelcome cells that may cause disease.

To classify blood types, scientists employ two types of antigens

- Antigens of the ABO blood group
- Antigens of the Rh-family

Antigens and antibodies are important components of the immune system's defensive system. Antibodies are produced by white blood cells. If an antigen is considered a foreign object, these antibodies will attack it. When a person requires a blood transfusion, it is critical to match blood types.

If a person obtains red blood cells that have antigens that are not previously present in their system, their body will reject and attack the new red blood cells, according to the American Red Cross. This can result in a severe and potentially fatal reaction.[6]

DIFFERENT TYPES OF BLOOD GROUP SYSTEMS

The designs of the diverse blood group and their antigens have been concentrated broadly, and an abundance of data is accessible especially since the turn of events of sub-atomic hereditary strategies. Less is thought about the genuine capacity of the blood gatherings. The red cell is a perplexing design, and the red cell film contains many surface proteins that are moored to the layer or cross the lipid bilayer at least multiple times.[3]

A significant number of the proteins on the outer layer of the red cells are polymorphic and convey the diverse blood gatherings. The elements of a portion of the red cell film proteins have been distinguished, and different capacities have been derived from the designs of the protein. Studies on the invalid aggregates which happen in most blood group have added to the data. The ABO, H, I, P1 and P blood groups are starch structures on the red cell layer glycolipids and glycoproteins and less is thought about their capacity.[4,5]

Table 1: Depicting different blood group systems

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
Rehsus	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
Bombay Blood Group	hh	1	FUT1	19

TYPE OF BLOOD GROUP

The International Society of Blood Transfusion now lists 33 blood group systems that represent approximately 300 antigens. The majority of them have been sequenced and cloned. Except for XG and XK, which are X-borne, and MIC2, which is found on both X and Y chromosomes, the genes of these blood group systems are autosomal. Integral proteins with polymorphisms in amino acid sequence [e.g., rhesus (Rh), Kell], glycoproteins, or glycolipids are examples of antigens (e.g., ABO). Here are a few of the most important organisations.[7]

Abo System

The presence or absence of the antigens A and B, which are carried on the surface of red blood cells, determines the ABO blood group system, which classifies human blood based on the inherited features of red blood cells (erythrocytes). As a result, people can have blood types A, B, O, or AB. Karl Landsteiner, an Austrian immunologist, was the first to identify the A, B, and O blood groups in 1901. Antibodies against type B red cells are found in the serum (fluid) of blood containing red cells with type A antigen on their surface. When type B blood is transfused into people who have type A blood, the antibodies in the recipient's blood kill the red cells in the injected blood. Anti-A antibodies in type B blood will damage type A red cells in the same way. Unless there is incompatibility with another blood group system, type O blood can be injected into people who have type A, B, or O blood. Type AB blood recipients can receive type A, B, or O blood. Because any person over the age of 6 months has clinically significant anti-A and/or anti-B antibodies in their serum, ABO remains the most important in transfusion and transplantation. Serum from blood group A contains antibodies against blood group B and vice versa, whereas serum from blood group O contains no A/B antigen but both antibodies.[8,9]

The ABO group is exceptional in that at whatever point the A or B antigens are absent on the red cells, the comparing immunizer is available in the plasma. Anti-A and Anti-B alloagglutinins are consequently regularly alluded to as being 'normally happening'. ABO gathering can consequently be performed by:

- Typing the red cells for the presence or nonappearance of the A or potentially B antigens. This is known as forward gathering.
- Testing the serum for the presence or nonattendance ofagainst An and additionally hostile to B antibodies. This is known as converse gathering.
- The forward and invert gathering results should correspond; allude to Landsteiner's standard.[6,7]

It ought to be noticed that the anti A,B created by a gathering O individual is unique in relation to anti A+B, which is a combination of anti A from one basis and anti B from another source. Anti A,B distinguished in group O people is an immunizer that will respond with group A and group B cells. The monoclonal anti A, B reagents accessible economically will distinguish the powerless A gathering Ax.[8]

BIO SYNTHESIS OF ABO ANTIGEN

The ABO red cell antigens communicated on the red cells are subject to the presence of both the H gene, and the ABO gene. The loci for the H and ABO gene are not connected in spite of the fact that they are connected and they are in this way two discrete blood groupgroups. The H, An and B gene don't code straightforwardly for red cell antigens,

yet for chemicals known as transferases.[9]

The H-transferase adds the sugar L-fucose to a forerunner substrate, which is a sugar chain as of now present on the red cell layer. Whenever this has been performed, the A-and B-transferases can act. The A-transferase adds another sugar called N-acetyl-D-galactosamine, which results in the statement of A antigen on the red cells. Essentially, the B-transferase adds the sugar D-galactose and the cells then, at that point, likewise express B antigen.[10]

These red cells type as gathering AB. Gathering A antigen is communicated when H and A transferases are the two compounds present; group B antigen is communicated at the point when the H and B transferases are the proteins present, and ingroup O just H transferase is available.[11]

The declaration of A, B or AB antigens brings about a family member concealing of the H antigen. Subsequently, A1, B or A1B cells express just little amounts of H. The A2 allele is less successful than the A1 allele in covering the H determinant, and A2 cells accordingly have significantly more H antigen and less A antigen than do A1 cells.[12]

The O allele in twofold portion prompts the outflow of H particularity alone, bringing about group O people having plentiful H antigen. The measure of H antigen that is available in red cells of various gatherings, from left or right in diminishing request, is as follows:

Most H antigen: O → Weak A → A2 → A2B → B → A1 → A1B → least H antigen.

RH SYSTEM

Rh blood group system, a technique for defining blood groups based on the presence or absence of the Rh antigen, also known as the Rh factor, on red blood cell cell membranes (erythrocytes). The name Rh comes from the fact that the basic test for identifying the presence of the Rh antigen in human blood uses the blood of rhesus monkeys. Karl Landsteiner and A.S. Weiner discovered the Rh blood group system in 1940. Since then, other different Rh antigens have been discovered, but the first and most frequent, RhD, generates the most severe immunological response and is the key determinant of the Rh characteristic. Because these immunological antibodies are immunoglobulin G (IgG), they can cross the placenta. Anti-D Ig prophylaxis against Rh immunisation is provided to Rh-negative moms who have given birth to Rh-positive children. If Rh-positive blood is donated in transfusion, the Rh antigen poses a risk to the Rh-negative person who lacks the antigen. Although adverse consequences may not appear the first time Rh-incompatible blood is administered, the immune system responds by generating anti-Rh antibodies in response to the foreign Rh antigen.[10] If Rh-positive blood is given again after the antibodies have formed, the foreign red blood cells will be attacked, leading them to clump together, or agglutinate. Hemolysis, or the destruction of red blood cells, results in significant disease and, in extreme cases, death. When the mother is Rh-negative and the father is Rh-positive, the Rh-positive offspring of Rh-incompatible parents face a similar risk during pregnancy. Unless the mother has developed anti-Rh antibodies as a result of an incompatible blood transfusion, the first child of such parents is normally unaffected. However, a little amount of the foetus' blood may reach the mother's system during labour. Anti-Rh antibodies are then produced by the mother, which attack any Rh-incompatible foetus in subsequent pregnancies. This results in erythroblastosis fetalis, or newborn hemolytic illness, which can be fatal to the foetus or infant soon after birth. If there is Rh-incompatibility, the sickness can be prevented by immunising the mother with Rh immunoglobulin after the birth of her firstborn. Before the mother's immune system can form antibodies, the Rh vaccine eliminates any foetal blood cells.[11,12]

The revelation of the Rh group via Landsteiner and Wiener in 1940, along with crafted by Levine and Stetson in 1939, proclaimed the best revelation in the blood gathering field since Landsteiner portrayed the ABO group in 1900. In 1939 Levine and Stetson portrayed how the mother of a stillborn embryo experienced an extreme haemolytic response when bonded with her better half's blood.[13] The mother, who clearly did not have some 'new' antigen, probably been vaccinated by her hatchling that had this antigen, having acquired it from the dad. At the point when the ABO viable spouse's blood was bonded, the maternal immune response responded with this equivalent antigen on his red cells. In 1940 Landsteiner and Wiener, having vaccinated bunnies with the blood of a rhesus monkey (Macaques mulatta), found that the subsequent antibodies agglutinated not just the monkey red cells yet additionally the red cells of around 85% of white individuals.[14,15] Later work, nonetheless, showed that the red cell antigens recognized by the human-inferred immune response and the creature immunizer were not indistinguishable and had a place with two distinct blood groupgroups. The blood group groups identified by the human-

inferred antibodies is currently known as Rh (not Rhesus) furthermore, the antigen is called D. The antigen initially depicted via Landsteiner and Stetson is LW in the LW blood group groups. The two groups are serologically, biochemically and hereditarily not quite the same as each other. The locus for the Rh gene is on chromosome 1 and is connected to the quality for elliptocytosis. The position for LW is on chromosome 19. Rhesus-group is the second most significant blood groupgroups after ABO. At present, the Rh-group comprises of 50 characterized blood group antigens out of which just five are significant. RBC surface of an individual might have a Rh factor or immunogenic D-antigen. As needs be, the status is demonstrated as either Rh-positive (D-antigen present) or Rh-negative (D-antigen missing).[16,17]

Rather than the ABO group, against Rh antibodies are, regularly, not present in the blood of people with D-antagonistic RBCs, except if the circulatory arrangement of these people has been presented to D-positive RBCs. These insusceptible antibodies are immunoglobulin G (IgG) in nature and subsequently, can cross the placenta. Prophylaxis is given against Rh vaccination utilizing hostile to D Ig for pregnant Rh-negative moms who have brought forth Rh-positive youngster.[18]

RH GENETICS AND INHERITANCE

Most people are either D+ or D–and the articulation or non-attendance of the D antigen on the red cells results from the presence or nonappearance of a RHD quality. A D+ individual may acquire two RHD genes, one from each parent (homozygous) or on the other hand one RHD quality from one or the other parent (hemizygous). The two sets of contradictory antigens C and c, and E and e are constrained by the different RHCE genes. The RHD and RHCE alleles are acquired as a quality complex or haplotype.[19]

Rh antigen

The Rh antigens are coded for by the RHD and RHCE genes, every one of which delivers a different protein that is embedded into the red cell layer. The RhD protein crosses the red cell layer multiple times, bringing about six extracellular spaces. In spite of many examinations, the specific capacity of the Rh proteins inside the red cell layer is obscure, yet their construction proposes a transmembrane carrier work. The elements of the RhD and RhCcEe proteins seem comparative. In instances of the extremely uncommon sort Rhnull, the shortfall of the Rh proteins has shown that the red cells are unusual morphologically and people regularly experience the ill effects of some level of haemolytic paleness as the red cells are strange fit.[20]

- ***H Antigen***

H-antigen is the precursor to the antigens that make up the ABO blood groups. It can be found in all RBCs, regardless of the ABO system. Homozygous for the H gene (HH), people with the unusual Bombay phenotype do not express H-antigen on their RBCs. Because H-antigen functions as a precursor, its absence indicates that antigen A and B are not present. Individuals, on the other hand, create isoantibodies to the H-antigen as well as antigens A and B.[13]

- ***Kidd System***

Kidd antigen (also known as Jk antigen) is a glycoprotein found on the surface of red blood cells (RBCs) that functions as a urea transporter in RBCs and renal endothelial cells. Kidd antibodies are uncommon, although they can result in serious transfusion responses. These antigens are identified by reactions to an anti-Jka antibody found in the serum of Mrs. Kidd, who gave birth to a child with HDFN. Jka was the first antigen discovered by the Kidd blood group system, and two more antigens, Jkb and Jk3, were discovered later. It conducted an automated investigation of blood groups in a north Indian donor community and discovered that the most prevalent blood groups were B, O, A, and AB, with 94.4 percent of the population being Rh-positive. The most common phenotypes in minor blood groups were Le (ab) for Lewis, Fy(a+b+) for Duffy, Jk(a+b+) for Kidd, and M+N+ for MNS system.[14]

The urea transporter in red blood cells (RBCs) is the Kidd (JK) glycoprotein. Its location in the membrane allows it to swiftly transfer urea into and out of RBCs while maintaining the RBC's osmotic stability and shape. The Kidd glycoprotein is also produced in the kidney, where it allows the kidney to accumulate a large level of urea, which is required for the kidney to produce concentrated urine. People who lack the Kidd glycoprotein are unable to concentrate urine to its maximum capacity, but they are otherwise healthy, and their RBCs have a normal shape and lifespan. Kidd antigen-specific antibodies are a common cause of delayed hemolytic transfusion events. Hemolytic disease of the newborn (HDN) is also caused by anti-Kidd antibodies. The severity of the condition varies, but it is usually moderate.[15,16]

- ***Duff System***

Duffy-antigen was discovered in a patient with haemophilia named Duffy. It's also known as Fy glycoprotein, and it's found on RBCs' surfaces. It's a nonspecific chemokine receptor that also serves as a receptor for the human malaria parasite Plamodium vivax. The Duffy glycoprotein antigens Fya and Fyb can result in four different phenotypes: Fy(a+b), Fy(a+b+), Fy(ab+), and Fy(ab). HTR can be caused by antibodies of the IgG class. Variations in the DARC gene, which encodes the chemokine receptor protein present on the surfaces of Duffy-expressing cells, give rise to Duffy antigens.[17] Antibodies to Duffy antigens Fy3 through Fy5 were found in the early 1970s, while antibodies to another antigen, Fy6, were discovered the following decade. Variations in the Fya and Fyb epitopes, which are parts of antigens capable of activating immunological responses, are represented by the Fy3 through Fy6 antigens. Purkinje cells in the brain, as well as cells in the colon, spleen, and thyroid gland, have been discovered to have Duffy antigens. Antibodies to the Duffy antigens have been linked to erythroblastosis fetalis and transfusion responses.[18, 19]

- ***Kell System***

After the ABO and Rh systems, these erythrocyte antigens are the third most potent immunogenic antigens, and they are characterised by an immunological antibody called anti-K. Mrs. Kellacher's serum was the first to notice it. She developed hemolytic responses after reacting to her newborn's erythrocytes. There have been 25 Kell antigens found since then. Anti-K antibody causes haemolytic transfusion responses and severe hemolytic illness of the foetus and newborn (HDFN) (HTR).[20]

There are a total of 25 Kell antigens, which are all encoded by the KEL gene. K and k, which encode the K (Kell) and k (Cellano) antigens, are the two major, codominant alleles of the KEL gene. The k antigen is widespread, with more than 90% of blacks and whites carrying it. Different antigens are produced by polymorphisms in the KEL gene, including the Jsa and Jsb antigens. The Jsb antigen is found in 100% of white people and 80% of black people. Kpa (Penney) and Kpb (Kelly) are two more Kell antigens. Kell antigens are generally found on the surfaces of red blood cells and connect with a protein called Kx. McLeod syndrome is caused by a lack of the Kx protein in some persons. Acanthocytosis (thorny projections on red blood cells) and low Kell antigen expression are two symptoms of this condition. These anomalies frequently result in muscular and nerve function problems, which present as slowed movement, emotional distress, and loss of reflexes.[21, 22]

- ***Lutheran System***

Lutheran blood group system, a technique for classifying human blood based on the presence of Lutheran antigens on red blood cell surfaces. There are 19 known Lutheran antigens, all of which are caused by mutations in the BCAM gene (basal cell adhesion molecule). The system is based on the expression of two codominant alleles, Lua and Lub, which are referred to as Lua and Lub, respectively. The Auberger antigens, Aua and Aub, were long assumed to constitute an unique blood group, but were eventually discovered to be Lutheran antigens resulting from mutations in the BCAM gene.[23]

Within populations, the traits Lu(a+b) and Lu(a+b+) are found at different frequency. In all groups, the Lu(ab+) phenotype is the most prevalent, whereas the Lu(ab) phenotype is extremely rare. Lua is rarely the cause of erythroblastosis fetalis or transfusion responses, despite its presence in the womb. The Lutheran system is made up of four pairs of allelic antigens that each indicate a single amino acid mutation in the Lutheran glycoprotein on chromosome 19. Antibodies to this blood group are uncommon and usually aren't regarded clinically important.[24]

- ***Mns Blood Group***

MNSs blood group system, a classification of human blood based on the presence of numerous chemicals on the surfaces of red blood cells known as M, N, S, and s antigens. This system, initially discovered in 1927, has a wide range of phenotypes and is useful in genetic and anthropological research on human populations. The MNSs blood group system has about 40 antigens. GYPA and GYPB are two highly polymorphic (variable) genes that encode these antigens (glycophorin A and B, respectively). The system is made up of two pairs of codominant alleles, M and N (first discovered in 1927) and S and s. (identified 1947 and 1951, respectively).

In most populations, the alleles M and N are dispersed in almost equal numbers. The S and s alleles, on the other hand, have different frequencies, with the S allele being found in about 55% of whites and 30% of blacks, and the s allele being found in roughly 90% of people in both groups.[25,26]

SsU, En(a), and Mk are examples of phenotypes in the MNSs antigen system caused by deletion mutations in the GYPA and GYPB genes. Genetic recombination (the exchange of genetic material

across genes) of GYPA and GYPB produces antigens such as He (Henshaw, 1951), Dantu, Sta (Stone), and Mia (Miltenberger). Incompatibility reactions are rare when antibodies to the M and N antigens are present. Antibodies to S, s, and a number of additional antigens, including Ena and Mia, can result in transfusion responses and foetal erythroblastosis. Glycophorin A and Glycophorin B are the two genes that make up the MNS antigen system, which was first reported by Landsteiner and Levine in 1927. The blood group is determined by an autosomal locus on chromosome 4 and a pair of co-dominant alleles known as LM and LN. Anti-M and anti-N antibodies are IgM antibodies that are only rarely linked to transfusion responses.[27,28]

- ***Bombay Blood Group***

Dr. Y M Bhende originally discovered the Bombay blood group in Mumbai (formerly Bombay) in 1952. The hh blood type is found in one out of every four million people. It is more common in South Asia; in India, one in every 7,600 to 10,000 children is born with this kind. An Antigen H blood test is required to check for hh blood. The hh blood group is sometimes confused with the O blood group. Antigen H is present in the O group, whereas it is absent in the hh group. Antigen H deficiency does not necessarily indicate a lack of immunity or an increased risk of disease. Based on their health index, their haemoglobin, platelets, white blood cells, and red blood cells counts are similar to those of others. They do, however, have issues with blood transfusions due to their rarity.[29]

"The persons with Bombay blood group can only be transfused autologous blood or blood from individuals of Bombay hh phenotype exclusively, which is exceedingly rare," according to a 2015 study published in the Asian Journal of Transfusion Science. If they receive blood from the A, B, AB, or O blood groups, they may be rejected. The hh blood group, on the other hand, can donate blood to ABO blood types. Over 350 donors for the Bombay blood group are listed on an unauthorised register in India. "However, there are only 30 active donors available at any given moment," said Vinay Shetty of the Think Foundation, an NGO. This group is rarely stored in blood banks, owing to its rarity and the 35-42 day shelf life of blood. As a result, anytime a Bombay blood group patient is needed, a donor is needed immediately. It is sometimes necessary to construct facilities for moving donated blood from one city to another. A patient in Kota received hh blood from a Pune based donor two weeks ago. The blood was flown to Jaipur and then transported by road to the Kota hospital.[30]

- ***Golden Blood Group***

The golden blood type, also known as Rh null, is characterised by the absence of Rh antigens (proteins) on the red blood cell surface (RBC). This is the world's rarest blood group, with fewer than 50 people worldwide carrying it. It was discovered in Aboriginal Australians for the first time. The problem with the golden blood group is that Rh null blood donations are extremely rare and difficult to come by. If a Rh null individual requires blood, they must rely on a small network of frequent Rh null donors all over the world. There are only nine active donors for this blood group on the planet. This makes it the most valuable blood type on the planet, hence the name golden blood. Antigens are proteins found on the surface of our red blood cells. We have one of four blood types: A, B, O, or AB, depending on the antigen present. The presence or absence of the 'Rh-D' factor on the cells determines whether the ABO system is Rh-positive or Rh-negative.[31]

Individuals with the golden blood group lack all of the Rh antigens, whereas those with the Rh-negative blood type lack only the RhD antigen. Genetic mutation appears to be the cause of the golden blood group (spontaneous change in the gene). Mutations in the RHAG gene, which codes for the Rh-associated glycoprotein, are common. The Rh antigens must be directed to the RBC membrane by this protein. The RHAG mutation is frequently linked to a condition known as hereditary stomatocytosis. Long-term, moderate hemolytic anaemia and accelerated RBC breakdown can occur in these people. The Rh-null phenotype can also be present in people who are born with certain anemias.[32]

PRACTICING DONE IN BLOOD GROUPING:

There are disagreements over the optimal procedure for obtaining blood in both elective and emergency situations: (a) In elective surgery patients, this can be accomplished by routinely requesting grouping and cross-matching. In surgeries where blood loss is not expected to be large, many scientific studies questioned the value of preoperative blood preparation.[20,21] (b) Blood can be ordered without a complete battery of tests.[19] ABO-Rh type alone ensures a 99.8% chance of a successful transfusion. Antibody screening boosts the safety margin to 99.94%, and a cross-match boosts the compatibility to 99.95%. There is a chance of missing antigens on donor cells if cross-matching isn't done, although

this isn't a big deal in clinical practise. As a result, only "screening and typing" should be done. Other methods include "type and partial cross-match," which includes the immediate phase of cross-match; "type and uncross match," which is performed in emergency situations when time for these procedures is limited; "type O Rh-negative uncross match," which is performed in emergency situations when time for these procedures is limited; and "type and uncross match," which is performed for those recipients who have never been transfused before, the chance of detection of antibody with each cross-match is 1:1000; and "type O Rh-negative uncross" n the latter case, type O Rh-negative packed RBCs, often known as universal donors, can be employed since they contain very little hemolytic anti-A/anti-B antibodies against the recipient RBCs.[33]

FORENSIC IMPORTANCE

Blood grouping is one of the most important aspects in the term of forensic investigation because the blood plays a very crucial role when it comes to individualization and identification. It is one of the most reliable source and one of the most common evidence which found on the crime scene. The condition in which the blood is present or encountered on the crime scene may vary. It helps in the identification of the person with respect to its height in the case of the spattering of the blood or blood spatter analysis. It also link suspects to the crime scene and also narrows down the investigation process.[34]

In blood analysis the presence of any kind of the foreign matter can also be detected by the performing certain test which may related to the condition of the victim and also to the intension of the suspect such as murder etc. it is also the individual in nature which means the list of the suspect can be narrowed down if the investigator properly handle the evidences and it also leads to free the innocent. It also play a very crucial role to link the evidence to the crime scene and crime scene to the suspect and the suspect to the victim. It also help in linking the crime scene i.e primary to the secondary.[35]

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