JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR) An International Scholarly Open Access, Peer-reviewed, Refereed Journal

Analysis of Blood and Blood Product Needs and Usage in a Tertiary Care Setting: A Comparative Study at NIMS Hospital in Jaipur, Rajasthan.

*Kumar Vivek¹, Anjali Goswami², Pandeep Kaur³, Davood UB⁴
¹PhD Scholor, ²PhD Scholor, ³Assistant Professor, Assistant Professor NIMS UNIVERSITY JAIPUR, RAJASTHAN

Abstract: Blood, comprising cellular components and plasma, plays a crucial role in sustaining life by supplying oxygen and nutrients to various body parts. The transfusion of blood and its components is an integral aspect of healthcare, but indiscriminate use can burden both patients and blood banks. This study aims to assess the patterns of blood and blood product requirement and utilization at NIMS Hospital in Jaipur, Rajasthan.

Materials and Methods: Conducted as a retrospective study over a four month period from January 2023 to April 2023, this research gathered essential data from blood centre registers and computers.

Results: Out of a total of 920 blood units utilized, Packed Red Blood Cells (PRBCs) were the most commonly used, followed by Whole Blood, Fresh Frozen Plasma (FFP), with platelet concentrates (Random Donor Platelets) being the least utilized. The Surgical Intensive Care Unit (SICU) emerged as the major beneficiary of the blood supply.

Conclusion: The study advocates for the formulation of stringent guidelines for transfusion practices to enhance the appropriate use of this precious resource. Periodic evaluations of utilization patterns and demands for various blood products are essential for maintaining an optimal blood stock. Implementing such measures can contribute to the judicious use of blood and its products, reducing unnecessary burdens on both patients and the blood bank.

Keywords: Blood transfusion, Blood utilization, Platelet concentrates, Fresh Frozen Plasma (FFP), Packed Red Blood Cells (PRBCs), Transfusion guidelines,

I. INTRODUCTION

Blood is a specialized bodily fluid that supplies essential substances and nutrients and removes metabolic waste products from the cells. It is composed of cells and plasma. The cellular components include red blood cells, white blood cells and platelets. Plas ma contains coagulation Factors. Blood is essential for human survival [1-8]. Until now there is no effective substitute for blood. Hence, transfusion of donated blood is the main stay of treatment in variety of medical/clinical conditions. Blood transfusion was first performed successfully by James Blundell in 1818 [1]. Blood component therapy has gained much of the interest in recent years because of its merits over whole blood transfusion like, it reduces volume overload on patient and has greater shelf life, better patient management [7]. Component therapy was introduced between 1950 and 1960s [7] to maximize the benefits of all components present in the whole blood [8-16]. Inappropriate transfusion practices can lead to serious consequences for recipients including transmission of infectious agents [16]. In developing countries there are limited resources of blood and increasing demand, hence it is necessary to make an efficient use of blood [17]. As blood is a scarce resource, clinician should weigh the risks of transfusion against risks of not transfusion often in both developed and developing countries [6,7]. Evaluation of pattern of blood component usage, its demand and good audit management is needed to ensure appropriate utilization of precious resource.

HISTORY OF DISCOVERIES OF THE BLOOD LANDSTEINER AND BLOOD TYPES

The man who discovered some of the human blood types that we know today was an Austrian, Karl Landsteiner. His work was influenced by an article on blood typing in goats, which was written by Paul Ehrlich and appeared in the 'Berliner klinische Wechenschrift' in 1900.

Landsteiner discovered the common blood types A, B and O (which he referred to as A, B and C) in 1901, and Adriano Sturli and Alfred von Decastello – who were working under Landsteiner – discovered type AB a year later in 1902. Landsteiner was awarded the 1930 Nobel Prize in Physiology or Medicine for his work [18].

OTTENBERG AND OTHERS

Six years after Landsteiner's discovery, in 1907, an American doctor named Reuben Ottenberg successfully transfused blood between two people at Mount Sinai Hospital in New York. He was the first person to record pre-transfusion testing for blood

compatibility in a clinical setting, although he remarked later that the testing "was only brought in incidentally in a footnote", and concluded that he probably "should have made a separate article".

Ottenberg made several notable discoveries over the next 50 years. His work led to the knowledge that people with type O blood are 'universal donors', which means that their blood will be accepted by people with any of the four ABO system blood groups [19]. Testing blood types made transfusion much safer, and it got steadily more popular. Yet some recipients were still undergoing transfusion reactions, suggesting that an important part of the puzzle was missing. In 1940 this was revealed as Rh factor, which was discovered by Landsteiner and Alexander Weiner in tests on rhesus monkeys (hence the label 'Rh', for

Rhesus). Whether you test positive or not for the Rh D antigen determines whether you have a positive (e.g. A+) or negative (e.g. AB-) blood type.

ABO and Rh are still the most recognised systems. The NHS website, for example, states that there are eight blood types (A+, A-, B+, B-, O+, O-, AB+ and AB-). This isn't strictly true, but it's all that most people will need to know.

BLOOD PRODUCTES & COMPONENTS

THE HISTORY OF BLOOD TRANSFUSION

It was in 1628 that the English physician William Harvey discovered the circulation of blood and soon after the first known blood transfusion was attempted. However, only in 1665 was the first recorded succesfull blood transfusion performed: physician Richard Lower kept dogs alive by transfusion of blood from other dogs. It was only in 1818 that the first succesful human to human blood transfusion saves the life of a woman suffering from profound postpartum hemorhage. The caring obstetrician, James Blundell from the United Kingdom, performed 10 transfusions of which only 5 were of benefit to the receiving patients.

In 1900, Karl Landsteiner, an Austrian physician, discovered the human blood groups for which he received the Nobel Prize for Medicine and this led to the discovery of the importance of crossmatching in 1907. The subsequently implemented crossmatching before transfusion prevented a substantial amount of acute hemolytic transfusion reactions. In 1914 were the first long-term anticoagulants developed and one of these, sodium citrate, was used a year later by Richard Lewisohn from Mt. Sinai Hospital in New York to change the practice of transfusion from direct (patient-to-patient) to indirect (patient-blood bank-patient). After the Second World War, Edwin Cohn from Harvard Medical School developed a system that enabled blood component therapy. The different blood components are described below.

RED BLOOD CELLS (ERYTHROCYTES)

In the late 1970s, both the new anticoagulant CPDA-1 as well as the first red blood cell (RBC) additive solution, salineadenineglucose (SAG) are developed. In 1981, the same researchers that developed SAG added mannitol to help protect the RBC membrane and reduce hemolysis. This

New solution was named SAGM and nowadays SAGM is the most widely used RBC additive solution. Of note, SAGM has not been licensed by the Food and Drug Administration (FDA), and hence is not used in the Unites States of America (USA). After having observed that a lower concentration of leukocytes led to less adverse effects, leukoreduction by means of filtration after collection is common practice in The Netherlands now. RBC volume is approximately 280 ml and the haematocrit ranges from 50% to 65%. One unit of RBCs transfused to an adult should in theory raise the haemoglobin concentration by 0.5 mmol/L. In the Netherlands, a bag of RBCs can be stored for up to 35 days at 2-6 °C.

FRESH FROZEN PLASMA

The first plasma transfusion in animals took place in the 1870s, when Bowditch and Luciani injected serum from sheep into frogs in their search for a blood substitute [25-27]. The first plasma transfusion in humans was to treat the Spanish flu in [28]. By the late 1920s and early 1930s, plasma became routinely used in hospitals [25,27,29]. Initially, fresh plasma was used, but researchers proved that both dried and frozen plasma were just as effective and easier to store [25,27,29,30]. During World War II, the United States Army used dried plasma as a blood substitute because of its easy storage [25,31]. However, it soon proved that trauma victims needed oxygen-carrying capacity and whole blood quickly replaced dried plasma. [25,31]. After World War II, the indications for a Fresh Frozen Plasma (FFP) transfusion was gradually extended to sepsis, burns, nutritional deficiencies, nephritic syndrome, sickle cell anemia and childhood acute lymphoblastic leukemia (ALL)25,32-36.

In 1964, the first randomized controlled trial on FFP transfusion was published37 followed. The only indications for which an FFP transfusion is indicated according to the current guidelines nowadays are single coagulation factor deficiencies for which no virussafe fractionated product is available, multiple coagulation factor deficiencies (e.g. diffuse intravascular clotting, DIC) in the presence of severe bleeding, thrombotic thrombocytopenic purpura (TTP), reversal of warfarin effect in the presence of severe bleeding and surgical bleeding and massive transfusion38.

Nowadays, Omniplasma is used instead of FFP in the Netherlands. Omniplasma is manufactured by thawing and pooling multiple units of single donor FFP. The advantage of Omniplasma is that it is a pooled product of 600-1200 donations which ensures adequate levels of clotting factors and the dilution of the possible presence of antibodies, thereby reducing the risk of transfusion-related acute lung injury. One bag of Omniplasma contains approximately 310 ml and can be stored for up to 4 years at -18 °C in the dark.

PLATELETS (THROMBOCYTES)

The first use of platelet transfusions was described in the 1950s to reduce mortality from haemorrhage in patients with acute leukaemia[39]. The use of platelets has steadily grown since then. The change from glass bottles to the disposable plastic bag sets for the collection of blood we still use nowadays was an important development, since it made collection and preparation of platelets within a closed system possible. This reduced the risk of bacterial contamination and facilitated the implementation of a simple, two-step centrifugation platelet preparation protocol. In the 1970s, investigators began removing leucocyte-rich and platelet-rich buffy coats from red-cell concentrates, to use the white cells for interferon production and to reduce leucocyte-related transfusion side effects. The regular use of this procedure led to the development of a novel whole-blood procedure for the preparation of platelet concentrates, named the buffy coat method, which we still use in the Netherlands. First, whole blood is spun which leads to the sedimentation of all cells. The thin layer of white blood cells and platelets is called the buffy coat and is removed from the other blood cells. Subsequently, five buffy coats of the same ABO/Rh group are collected, pooled, and diluted in plasma or in a

crystalloid solution. The pooled buffy coats are gently centrifuged and the platelet-rich supernatant is collected. One bag of platelets contains approximately 330 ml, the amount of platelets is at least 250×109 /ml and the storage solution is Platelet Additive Solution type III (PAS-III). A bag of platelets can be stored for up to 7 days, when kept at 20-24 °C on a shaking device.

METHODOLOGY

The transfusion data information so obtained from the documents was fed into Microsoft excel sheet for future analysis. The data pertaining to the number of /packed red cells/fresh frozen plasma/random donor platelet concentrates/single donor aphaeresis platelet concentrates requested as well as cross match/issue data was collected and analyzed.

Blood component preparation was developed in 1960 to separate blood products from one unit whole blood by specialized equipment called as refrigerated centrifuge [20]. Preparing only Packed Red Blood Cells (PRBCs) and fresh frozen plasma (FFP) is by single-step heavy spin centrifugation; however preparing platelet concentrates (PLTCs), PRBCs and FFP is by two step centrifugation. The two main procedures of preparing PLTC are either by platelet-rich plasma (PRP) method or Buffy Coat (BC) method.

However, PRP method is simple, easily done manually and comparatively cheaper and also platelet and plasma yield is good. BC method is a better method but complicated if done manually and hence needs automation. The main components are PRBC, PLTC or random donor platelet (RDP), FFP, cryoprecipitate, cryo poor plasma (CPP) and Plasma fractionation products. The last are produced only at the pharmaceutical industries end.

There are a variety of blood products, pharmacologic agents, and procedures that can be utilized to treat anemia, thrombocytopenia, and bleeding disorders. Here is a brief overview of the products and services available.

PRODUCTS	DESCRIPTION
	Packed red blood cells (PRBCs) are made from a unit of whole blood by centrifugation and removal of most of the plasma, leaving a unit with a hematocrit of about 60%. One PRBC unit will raise the hematocrit of a standard adult patient by 3% (or about 1%/mL/kg in a child - 12%/25 kg with the standard 300 mL PRBC unit). PRBCs are used to replace red cell mass when tissue oxygenation is impaired by acute or chronic anemia.
	FFP contains all factors of the soluble coagulation system, including the labile factors V and VIII. FFP is indicated when a patient has MULTIPLE factor deficiencies and is BLEEDING. Note that FFP SHOULD NEVER be used as a plasma expander.
	Cryoprecipitate (cryo) contains a concentrated subset of FFP components including fibrinogen, factor VIII coagulant, vonWillebrand factor, and factor XIII. Cryoprecipitate is used for hypofibrinogenemia, von Willebrand disease, and in situations calling for a "fibrin glue." Cryo IS NOT just a concentrate of FFP. In fact, a unit of cryo contains only 40-50% of the coag factors found in a unit of FFP, but those factors are more concentrated in the cryo (less volume).
	A single platelet unit is derived from one whole blood unit collected. Platelets are stored at room temperature and CANNOT be frozen. They must be used in 5 days. Pooled platelets from multiple donors from whole blood collections are cheaper to produce but the exposure to the recipient increases.

TRANSFUSION IN THE CRITICALLY ILL – TWO SIDES OF THE MEDAL TRANSFUSION OF RED BLOOD CELLS

Anaemia is a frequently encountered problem in the critically ill patient. As a consequence, critically ill patients are frequently transfused, with up to 44% of patients receiving a blood transfusion. In sepsis, transfusion rates can even reach 73%. The obvious causes of anaemia in critically ill patients are a decreased production of red blood cells (e.g. by a poor nutritional status, the existence of co-morbidities, a reduced production of erythropoietin and a reduced iron availability) and an increased loss of red blood cells (e.g. increased blood loss due to surgical procedures or repeated phlebotomy or bleeding).

In addition to that, there are indications an increased clearance of RBCs also plays an important role that in inflammatory states. It is commonly thought that the clearance of red blood cells in inflammation is mediated in the spleen. This has, however, never been proven and other mechanisms of clearance may be implicated. These include an increased PS-exposure on the red blood cell membrane as a consequence of increased plasma concentrations of sphingomyelinase induced by tumor necrosis factor- α or direct production by bacteria[40,41], which then can act as an "eat-me" signal to circulating macrophages. Also, RBCs can be phagocytosed directly by increased expression of Band 3 on their membrane , an increase that is present in septic mice but absent in heathy mice. Inflammatory conditions induced by LPS or sepsis can also lead to a decreased deformability of the red vlood cell, which may eventually lead to the trapping of RBCs in the microvasculature. Also, trapping of RBCs in the microvasculature during inflammation may be mediated by adhesion of RBCs to the endothelium in vitro [42,43].

Since RBCs transport oxygen from the lungs to the tissues, it has since long been common practice to supplement red blood cells liberally in order to ensure an adequate supply of oxygen to the tissues. However, the Transfusion Requirements in Critical Care (TRICC) trial demonstrated that lower Hb levels were well tolerated in the critically ill40. Also, younger patients and the less severely ill transfused according to the restrictive transfusion strategy had an improved 30-day mortality rate compared with the patients in the liberal strategy group. Hereby, is an association between RBC transfusion and adverse outcome. In line with this, several

observational studies have reported an association between RBC transfusion and adverse outcome in the critically il [144].

In particular, an association between RBC transfusion and the development of acute lung injury [45] and acute kidney injury [46,50] is repeatedly found. TRALI (Transfusion- Related Acute Lung Injury) is a clinical diagnosis and defined as the onset of acute lung injury within 6 hours of blood transfusion without an additional risk factor for acute lung injury [51-53]. Also possible TRALI (which is TRALI in the presence of another risk factor) and delayed TRALI (which is TRALI within 6-72 hours after the blood transfusion) have been described as clinical entities [51-53]. TRALI incidence varies between 0.08–15.1% per patient transfused and 0.01–1.12% per product transfused [54,5]. TRALI occurs more frequently in the critically ill patient population [49,54,55].

Although the finding of an association between transfusion and organ injury has led to the adoption of a lower transfusion trigger in most Intensive Care Units (ICUs) [19,56], a large variance in transfusion practice remains [57]. As anemia is also associated with adverse outcome and the currently used low Hb transfusion trigger may already be too low for some patient populations on the ICU [58], this poses a challenge to both the treating physician as well as to the scientific community, calling for interventions which improve RBC transfusions and reduce the risk of associated organ failure. Of note, the mechanisms of the observed association between RBC transfusion and adverse outcome still remain unknown.

In animal studies, prolonged storage of the transfused red blood cells is clearly related with the development of lung injury [59-66]. Clinical data, however, are conflicting. There are observational studies showing association between transfusion with stored red blood cells and adverse outcome and increased mortality [45,67], whereas other studies not found an association between age of blood and outcome [49,68-70]. These differences may be explained by the heterogenous study populations and the fact that most patients received both fresh and stored red blood cell products. Recently, two large randomized controlled trials found no difference in clinically relevant outcome parameters between critically ill patients receiving fresh and those receiving stored red blood cell products [71,72]. However, these findings do not rule out that storage lesion exists.

Given that the association between transfusion and organ failure exists, the need for improved preparation and storage conditions remains.

Organ injury is thought to be caused by bioactive substances which accumulate during storage of cellular blood products. Bioactive lipids [54,59,64,65,73-76] and sCD40L [54,63,77,78] have been implicated as soluble mediators in TRALI, but other studies have not confirmed this association79. Recently, extracellular vesicles (EVs) have been proposed as the responsible mediators. EVs are small phospholipid vesicles released from most cell types. EVs facilitate intercellular exchange of receptors, ligands, signaling molecules, genetic information, etc. without direct cell-to-cell contact. High concentrations of RBC-derived EVs are present in the supernatant of RBC transfusion bags [80]. propagate thrombin generation and shorten clotting time in vitro [80-82]. These effects of EVs likely depend on storage time of the blood bags, because the concentration and thrombin-generating ability of EVs increases with storage duration [81,82]. Alternatively to a causative soluble factor, the RBC itself also undergoes changes during storage. For example, it loses Duffy antigen expression and thereby chemokine scavenging function. The Duffy antigen is a minor blood group antigen that binds a variety of inflammatory chemokines, thereby rendering these red blood cell bound chemokines inaccessible to circulating neutrophils83. Furthermore, when erythrocytes age they lose their deformability, which impedes their passage through the microcirculation of the organs84. The subsequent adherence may even be further augmented in inflammatory conditions, due to activated endothelium. In conclusion, despite the obvious benefits and optimalization of the storage process which has taken place, an association between RBC transfusion and adverse effects remains, which warrants further research into the mechanisms of this association in an effort to optimize RBC storage conditions.

TRANSFUSION OF FRESH FROZEN PLASMA

Substantial units of FFP are utilized in the ICU85,. In practice, FFP is transfused to correct abnormal coagulation tests to prevent bleeding. Studies show that the prevalence of coagulation abnormalities in critically ill patients is high: 30-66% of patients have an International Normalized Ratio (INR) of >1.5 or a prothrombin (PT) ratio of >1.587 [,88,] to 45% has a thrombocytopenia at some point during their ICU stay. The most common causes of a deranged coagulation are sepsis, multiple trauma, brain injury, major blood loss, liver disease, disseminated intravascular coagulation, use of vitamin K antagonists before ICU admission, vitamin K deficiency, renal failure, cardiac surgery and thrombotic micro-angiopathies.

Fresh frozen plasma effectively corrects multiple clotting factor deficiencies and guidelines recommend its use in severe bleeding, but also patients who have a coagulopathy, but lack signs of active bleeding receive a substantial amount of FFP [85].

Even when evidence that prophylactic administration prevents bleeding complications is absent inappropriate use of FFP is widespread. In the Netherlands, 80.000 FFP units are issued annually and most of these are transfused in the ICU [85]. Three misapprehensions are deemed responsible for this inappropriate use of FFP: physicians assume that an elevated PT or INR predicts an enhanced bleeding risk in patients undergoing an invasive procedure, that the pre-procedural FFP administration improves PT/INR values and that prophylactic FFP transfusion results in fewer bleeding complications1. But does an elevated PT/INR predict an enhanced bleeding risk? The coagulation system consists of three main components. The pro-coagulant elements include the endothe [14] lium, thrombocytes, individual coagulation factors and fibrinogen. The anti-coagulant system includes proteins C and S and antithrombin. The third component of coagulation is the fibrinolytic system. Most standard coagulation tests (platelet count, aPTT, PT, fibrinogen and d-dimer) only reflect a part of this complex system, and therafore fail to reflect the contemporary result of the balance between the three separate components.

Thereby, standard coagulation tests cannot reliably predict potential bleeding risk. In contrast to these conventional coagulation tests, rotational thromboelastography assesses both clot formation and degradation. The resulting thromboelastogram represents initiation of clot formation, fibrin formation and clot degradation. In critically ill patients suspected of a coagulopathy,

thromboelastography may improve identification of patients who have an increased bleeding risk and are in need of FFP transfusion. However, threshold values indicative of coagulopathy have not been validated for the critically ill.

Furthermore, does pre-procedural FFP administration improve PT/INR values and in general, restore coagulation ability? Only a few clinical trials have studied the efficacy of transfusion of FFP in critically ill patients with a coagulopathy, but these used different doses, did not assess the effect of FFP administration on occurrence of bleeding complications and included both bleeding and nonbleeding patients. In assessment of efficacy of FFP, the dose is of importance. An adequate dose of FFP will correct the PT/ INR, since it suppletes all clotting factors. However, since the PT/INR only reflects part of the coagulation system, this does not mean that an FFP transfusion effectively corrects coagulopathy. Altogether, evidence supporting the efficacy of FFP to correct coagulopathy in critically ill patients is scarce. Furthermore, no evidence exists that prohylactic transfsufion of FFP to critically ill patients with a coagulopathy can reduce bleeding complication. Of interest, epidemiological studies in trauma patients suggest that in trauma patients requiring a massive transfusion, resuscitation with a higher ratio of FFP to red blood cell units was associated with decreased mortality. Of note, this association was independent of the effect of FFP on correction of coagulopathy. As FFP is supposedly beneficial in bleeding because it corrects a deficiency in coagulation factors, this observation suggests another mechanism of action of FFP. Comparable to a RBC transfusion, related circulatory overload and an increased risk of infections. But most importantly, FFP has been linked to TRALI43,68.

More specifically, antibody-mediated TRALI, which is caused by the passive infusion of human leucocyte antigen (HLA) and human neutrophil antigen (HNA) antibodies in donor blood. These antibodies are found predominantly in blood from multiparous women as they have become sensitized during pregnancy by becoming exposed to the antigens of their fetus.

The risk of female plasma donation was confirmed in two studies in critically ill patients. The United Kingdom implemented a male donor fresh-frozen plasma transfusion policy and many countries followed. TRALI incidence has decreased significantly since then and two recent meta-Analyses reported that excluding female donors reduces plasma-related TRALI incidence by 73%. However, the policy of use of male plasma only has decreased TRALI, but has not completely abrogated this risk. In conclusion, despite its widespread use in the ICU we still lack knowledge on all the effects of an FFP transfusion. Knowledge on these effects is needed to weigh the benefits and risks of plasma transfusion in the critically ill.

TRANSFUSION OF PLATELETS

Trombocytopenia is a frequent finding in the critically ill. Approximately percent of adult patients admitted to an ICU have a trombocytopenia (platelet count less than 150 x 109 per L). There is a wide variety of causes of thrombocytopenia in the critically ill, of which sepsis, disseminated intravascular coagulation (DIC), massive blood loss, thrombotic microangiopathy, heparin-induced thrombocytopenia, immune thrombocytopenia and drug-induced thrombocytopenia are the most important.

Transfusion trigger for platelets in the critically ill has not been formulated. Transfusion triggers have been established based on evidence from randomized trials, and resulting guidelines, although not specifically designed for the critically ill patient population, recommend different transfusion triggers, ranging from 10×109 to 50×109 per L for prophylactic transfusions for different indications.

Although thrombocytopenia is a risk factor for adverse outcome, such as major bleeding, increased length of ICU-stay and death, studies show conflicting results on whether platelet transfusions improve or worsen surviva. Arguably, worsening of survival may be a confounder to disease severity. However, there may also be adverse effects of a platelet transfusion, which may outweigh the supposed benefits. These include transmission of infection, allergic reactions, TRALI42,49,53,68, transfusionrelated immunomodulation (TRIM) and venous thromboembolism. TRIM has been associated with delayed graft rejection, increased

www.jetir.org (ISSN-2349-5162)

cancer recurrence and higher susceptibility to nosocomial bacterial infections. Indeed, studies showed that platelet transfusion is associated with nosocomial infections in a variety of critically ill

patient populations. Therefore, the benefit of a platelet transfusion should be weighed against the risks and a transfusion trigger should be carefully determined. Whereas naive platelets have a natural life span of 8-12 days, those prepared for transfusion can be stored only for 2-7 days at 20-24 C with agitation. This primarily reduces the risk of bacterial growth and secondarily minimizes the "platelet storage lesion" (correlates with reduced in vivo recovery and survival as well as haemostatic activity after transfusion. Although aged platelets are clearly associated with the development of TRALI in animal models [54,61,63,73,75],, clinical studies show conflicting results [49,54,68-70].

MATERIALS

The study is conducted in tertiary care NIMS hospital-based blood bank, catering to all types of patients.

Parameters of performance indicators measured

Performance indicator (serology)-Parameter

1) No. of PATIENTS samples accepted

2) No. of units cross – matched

3) No. of units issued

4) Cross-match transfusion comparison; Whole blood, Packed red blood cells, Fresh frozen plasma, Random donor platelet.

Performance indicator Transfusion transmitted infection (TTI area) Parameter

1) No. of nonreactive unit; Human immunodeficiency virus (HIV), Hepatitis B surface antigen (HBsAg), Hepatitis C virus (HCV), Syphilis, Malaria.

2) No. of reactive unit; Human immunodeficiency virus (HIV), Hepatitis B surface antigen (HBsAg), Hepatitis C virus (HCV), Syphilis, Malaria.

Performance indicator (component area)

Parameter

No. of component prepared; Whole blood, Red cell, Platelets, Fresh frozen plasma.

LIST OF PARAMETERS USED IN THIS STUDY

1.	SEROLOGY AREA	BLOOD UNITS
А.	NUMBER OF BLOOD REQUESTED	995
B.	NUMBER OF CROSMATCH UNIT	950
C.	NUMBER OF ISSUED UNIT	920
	TOTAL COMPONENT PREPARED	920
Ι	WHOLE BLOOD	98
II	PRBC	565
III	FFP	280
IV	RDP	30

OBSERVATIONS

A total of 995 requests of blood components received in blood bank of NIMS hospital, NIMS University, from 1/1/2023 to 30/4/2023 are examine. This is represented in (table-1) graphically by using bar graph and pie chart.

TABLE-1. BLOOD COMPONENT WISH DISTRIBUTION.

Blood Components	WB	PRBC	FFP	RDP	TOTAL
OBSERVATION					
Components Request 120 565 280 30 995					
Components Issued	98	530	262	30	920



FIGURE-1; VIVEK KUMAR PHD BLOOD TRANSFUSION- Requirement and Utilization of Blood and Its Components in Nims Tertiary Care Hospital Jaipur. Raj. India

TABLE-2 .COMPARISON OF MOST UTILIZED BLOOD (PRBCs) IN NIMS TERTIARY CARE HOSPITAL, JAIPUR.

SN.	TYPE OF BLOOD GROUP	UTILIZATION OF BLOOD
1	A POSITIVE	214
2	B POSITIVE	362
3	AB POSITIVE	112
4	O POSITIVE	190
5	A NEGATIVE	09
6	B NEGATIVE	27
7	AB NEGATIVE	-
8	O NEGATIVE	06
Т	OTAL-	920



FIGURE-2; VIVEK KUMAR PHD BLOOD TRANSFUSION; Comparison of most utilized blood and its components in Nims Tertiary Care Hospital Jaipur. Raj. India.

TABLE-3. COMPARITIVE STUDY OF MOST UTILIZED BLOOD COMPONENT BETWEEN W.B Vs PRBC Vs FFP Vs. RDP IN NIMS TERTIARY CARE HOSPITAL JAIPUR.

S.N.	UTILIZATION OF COMPONENT	UTILIZATION OF BLOOD UNITES
1	W.B.	98
2	PRBC	530
3	FFP	262
4	RDP	30



FIGURE-3; VIVEK KUMAR PHD BLOOD TRANSFUSION; COMPARISION OF MOST UTILIZED BLOOD **COMPONENTS IN Nims Tertiary Care Hospital Jaipur. Raj. India.**

TABLE-4.COMPARISON OF UTILIZED BLOOD COMPONENTS BETWEEN MALE AND FEMALE. IN NIMS TERTIARY CARE HOSPITAL JAIPUR.

MALE		FEMALE		
535		385		
Series 1				



Male Female FIGURE-4; VIVEK KUMAR PHD BLOOD TRANSFUSION; COMPARISION OF UTILIZED BLOOD AND ITS COMPONENTS FOR MALE Vs FEMALE IN Nims Tertiary Care Hospital Jaipur. Raj. India. TABLE-5.COMPARISION OF VARIOUS WARDS OR UNITS FOR MOST UTILIZATION OF BLOOD AND BLOOD **COMPONENTS**

S.N.	Various wards	Blood Utilized	S.N.	Various wards	Blood Utilized
1.	OBS	56	14.	FMW	40
2.	MICU	99	15.	MSW	26
3.	SICU	199	16.	PEADIATRIC	3
4.	CTVS	124	17.	MMW	16
5.	URO	50	18.	GFW	20
6.	POW	39	19.	PICU	33
7.	FOW	10	20.	NICU	2
8.	MOW	22	21.	EMR	16
9.	NEURO	4	22.	DW	2

10.	OBG	20	23.	OD	3
11.	CCU	54	24.	FSW	48
13.	PSW	5			
Total		920 units			



FIGURE-5; Vivek Kumar Phd Blood Transfusion; Comparision Of Various Wards Or Units For Most Utilization Of Blood And Blood Components In Nims Tertiary Care Hospital Jaipur. Raj. India.

S.N.	Various Disease	Blood Utilized (units)	S.N.	Various disease	Blood Utilized (units)
1.	Anemia	389	18.	Surgery	116
2.	RTA	87	19.	CABG	63
3.	Hb low	28	20.	OT	40
4.	CKD,HTM,MHD	36	21.	CAD	7
5.	Bleeding	14	22.	Emergency	08
6.	Multiple	03	23.	RHD, SVM	06
7.	HTN	19	24.	MVR	09
8.	Dehydration	18	25.	Fever	03
9.	Shock	04	26.	Haematuria	03
10.	Bladder Disorder	03	27.	Abdominal Pain	04
11.	Renal Calculum	03	28.	PCNL	03
12.	IDD	03	29.	Prolapse	04
13.	Pregnancy	04	30.	LSCS	04
14.	Mayomactomy	08	31	COPD	08
15.	Accidental	06	32.	Ulcer	10
16.	Post Operative	03	33.	TAH	02
17.	Kidney Disorder	02			

TABLE-6; COMPARISION BETWEEN VARIOUS DISEASES FOR MOST UTILIZATION OF BLOOD AND ITS COMPONENTS



FIGURE-6; VIVEK KUMAR PHD BLOOD TRANSFUSION; COMPARISION BETWEEN VARIOUS DISEASES FOR MOST UTILIZATION OF BLOOD AND ITS COMPONENTS IN Nims Tertiary Care Hospital Jaipur. Raj. India.

DISCUSSION

The study was carried at blood centre of NIMS HOSPITAL, JAIPUR, RAJASTHAN from 01/01.2023 to 30/04.2023. The study includes performance indicators in blood bank in- Examination area. And Serological area.

ACCORDING TO EXAMINATION AREA.

This study is concludes that the no of requested components 995 and issued the components are 920 units another study Total blood units collections were 13,378. Units utilized were 12,555. Whole blood was the most utilized product followed by PRBC, FFP and least utilized product was platelet concentrates Giriyan SS, Chethana HD*, Sindhushree N, Agarwal A, Nirala NK and Bajpai RDepartment of Pathology, KIMS, Hubballi, Karnataka, India jan. 2015.

ACCORDING TO CROSS MATCH OR SEROLOGICAL AREA.

Present study concluded that the no of issue components are 920 units for 995 requests and PRBCs are issued 530units, ffp are 262, wb are 98 and RDPs 30 is less than of all components and another study is A total of 2434 blood transfusion requisitions were received by the Department of Transfusion Medicine for 3621 (49.69%) packed red cells (PRBC) units, 2140 (27.59%) fresh frozen plasma (FFP), 1923 (24.80%) random donor platelet concentrates (RDP), 15 (0.19%) whole blood units and 55 (0.56%) *Corresponding author: Dr. Daljit Kaur, Consultant, Department of Transfusion Medicine, Max Super specialty Hospital, Dehradun, India, at june 2016.

CONCLUSION

The Present study was carried out in the Department of IH&BT Nims Blood Center, COMPARETIVE STUDY OF REQUIRMENT & UTILIZATION OF BLOOD & BLOOD PRODUCTS IN TERTIARY CARE NIMS HOSPITAL JAIPUR, RAJASTHAN. The present study includes a total of 920 units of donor blood for recipient in the period of three months. According to performance indicators of blood bank.

1) Examination area

2) Serology area

3) 920 units of blood are collected for 995 units of blood according to request forms.

This study is a retrospective longitudinal study and the carried out requirement and utilization of blood and its components in tertiary care NIMS HOSPITAL, JAIPUR, RAJASTHAN.

ACKNOWLEDGMENT

I extend my heartfelt gratitude to all those who have contributed to the successful completion of this research project on the & quot; Analysis of Blood and Blood Product Needs and Usage in a Tertiary Care Setting: A Comparative Study at NIMS Hospital in Jaipur, Rajasthan."

First and foremost, I would like to express my sincere appreciation to the administration and staff at NIMS Hospital for granting permission to conduct this study and providing invaluable support throughout the research process. Their cooperation and willingness to share critical insights have been instrumental in the success of this project.

I am deeply indebted to the medical professionals, laboratory technicians, and support staff at NIMS Hospital, whose dedication and cooperation made data collection and analysis possible. Their commitment to advancing healthcare practices has been truly inspiring.

I would like to acknowledge my academic advisor Dr Pandeep Kaur and my co- advisor Dr Davood UB for their guidance, expertise, and unwavering support. Their insightful feedback and encouragement have been instrumental in shaping the direction of this research.

I am grateful to my peers and colleagues who have provided valuable input and feedback during the various stages of this study. Their constructive criticism and collaborative spirit have enriched the quality of the research.

Finally, I express my gratitude to my family and friend Ms. Anjali Goswami for their unwavering support, understanding, and encouragement throughout this academic endeavor. Their patience and belief in my abilities have been a constant source of motivation.

This research would not have been possible without the collective efforts of all those mentioned above. Thank you for being an integral part of this academic journey.

REFERENCE:

1. Mathew AS, Kurian SS, Sundaresan NP, Jayalekshmi, Roderigues FP, et al. (2014) Pattern of blood component utilization in a teaching hospital in South Kerala. Academic Medical Journal of India 2: 28-31.

2. Gaur SD, Negi G, Chauhan N, Kusum A, Khan S, et al. (2009) Utilization of blood and components in a tertiary care hospital. Indian J Hematol Blood Transfus 25: 91-95.

3. Venkatachalapathy TS, Subhashish D (2012) A prospective audit of blood transfusion requests in RL Jalappa hospital and research centre for blood and blood components. J Blood Lymph 2:106.

4. Ambroise MM, Ravichandran K, Ramdas A, Ganthimathy Sekhar (2015) A study of blood utilization in a tertiary care hospital in South India. J Nat Sci Biol Med 6: 106-110.

5. Joshi RA, Ajmera JR, Kulkarni SA, Bindu SR, Kulkarni SS (2014) Observational study in Utilization of blood and blood products at tertiary care centre. International Journal of Health Sciences and Research 4:38-47.

6. Alcantara CJ, Opina PA, Alcanatara MR (2015) Appropriateness of use of Blood products in tertiary Hospitals. International Blood Research and Reviews 3: 54-65.

7. Chowdhury FS, Siddiqui A, Islam K (2015) Use of blood and blood components in Dhaka medical college Hospital 26: 18-24.

8. Alcanatara YT, Alresheid A, Shammary S (2015) A comparative study on blood components utilization in selected Hospital-Blood banks in Hail, K.S.A. IOSR Journal of Nursing and Health Sciences 4: 28-33.

9. Pozo EA, Rosales PM, Almeida-Neto Cd, Remesar MC, Cortes AD, et al. (2015) A comprehensive protocol to evaluate the use of blood and its components in Latin America and the Caribbean. Rev Panam Salud Publica 37: 435-441.

10. Bansod P, Jethani N, Pachori G (2015) Clnical use of Blood and its components in tertiary health care centre in northwestern India. Int J Med Sci Public Health 4: 787-791.

11. Garg R, Aggrawal R (2013) An audit of the Blood and component transfusion requests and utilization pattern in a tertiary care hospital-Current trends. International. Journal of Drug Discovery and Medical Research 1: 82-85.

12. Gomathi G, Varghese R, Falleiro JJJ, Lakhani D, Garg S, et al. (2012) Audit of use of blood and its components in a tertiary care centre in South India. Asian Journal of Transfusion Medicine 6: 189-190.

13. Ahmed M, Save US (2016) Blood components Therapy in paediatric intensive care unit in tertiary care centre: An audit. International Journal of Contemporary Medical Research 3: 1506-1510.

14. Bhat WA, Aziz R, Ahmed CB, Ahmed IS (2012) Utility of Blood components in Paediatric patients. An Audit 16: 61-63.

15. Anshoo A, Saidunnisa B, Meghna C, Emadullah R (2013) Where does blood go? Study on transfusion practices in SAQR hospital, Ras AI Khaimah, UAE. International Journal of Science and Research 2: 56-59.

16. WHO (2009) Guidelines and Priniciples for safe blood transfusion practices pp: 25-34.

17. Mackroo RN (2009) Transfusion practice in clinical medicine. In: Compendium of Transfusion medicine, pp: 217-218.

18. Nobelprize.org's biography of Karl Landsteiner.

19. PBS's biography of Reuben Ottenberg .

20. Moog R. A new technology in blood collection: Multicomponent apheresis. In: Peterson BR, editor. New Developments Transfusion Research. New York: Nova Science Publishers, Inc; 2006. pp. 141–6.

21c. Jump up to:a b Blood Group A Suptypes, The Owen Foundation. Retrieved 1 July 2008.

22. Jump up^ Seltsam A, Hallensleben M, Kollmann A, Blasczyk R (2003). "The nature of diversity and diversification at the ABO locus". Blood. 102 (8): 3035–42. doi:10.1182/blood-2003-03-0955. PMID 12829588.

23. Jump up^ Ogasawara K; Bannai M; Saitou N; et al. (1996). "Extensive polymorphism of ABO blood group gene: three major lineages of the alleles for the common ABO phenotypes". Human Genetics. 97 (6): 777–83. doi:10.1007/BF02346189. PMID 8641696.

24. Jump up^ Shastry, S; Bhat, S (October 2010). "Imbalance in A2 and A2B phenotype frequency of ABO group in South India". Blood Transfusion. 8 (4): 267–270. doi:10.2450/2010.0147-09. PMC 2957492 . PMID 20967168.

25.plasma: the most commonly prescribed haemostatic agent. Journal of thrombosis and haemostasis: JTH 2013;11:1794-9.

26. Amberson WR. Blood substitutes. Biol Rev Camb Philos Soc 1937;12:48-86.

27. Strumia MMJ. The development of plasma preparations for transfusion. Annals of internal medicine 1941;15:80-8.

28. Hartman F. New methods for blood transfusion and serum therapy. J Am Med Assoc 1918;71:1658-9.

29. Strumia MMJ, Wagner JA, Monaghan JF. The intravenous use of serum and plasma. Fresh and Preserved. Ann Surg 1940;111.

30. Edwards FR, Kay J, Davie TB. Preparation and Use of Dried Plasma for Transfusion. British medical journal 1940;1:377-81.

31. Starr DP. Blood: an Epic History of Medicine and Commerce. 1st edn New York: Alfred A Knopf, 1998.

32. Nicholson P. Notes on the treatment of an unusual case of hemolytic Streptococcus septicemia. J Pediatr 1936;8:363-6.

33. Grant IC, Irons MG, Morris J, Forbes J. Subcutaneous plasma infusion in convalescent children. Va Med Mon 1918;1949:182-5.

34. Kaplan E, Lewis SR. The effect of human plasma transfusion on the fecal urobilinogen excretion on sickle cell anemia. Blood 1949;4:947-57.

35. Darte JM, Snelling CE, Donohue WL. The effect of plasma infusions in acute leukaemia in children. Can Med Assoc J 1952;66:576-8.

36. Stickler GB, McKenzie BF, Wakim KG, Burke EC. The effect of plasma transfusion and treatment with corticotropin on the electrophoretic patterns in serum and urine of children with the nephritic syndrome. The Journal of laboratory and clinical medicine 1956;47:392-402.

37. Trimble AS, Osborn JJ, Kerth WJ, Gerbode F. The Prophylactic Use of Fresh Frozen Plasma after Extracorporeal Circulation. The Journal of thoracic and cardiovascular surgery 1964;48:314-6.

38. O'Shaughnessy DF, Atterbury C, Bolton Maggs P, et al. Guidelines for the use of fresh-frozen plasma, cryoprecipitate and cryosupernatant. British journal of haematology 2004;126:11-28.

39. Freireich EJ. Supportive care for patients with blood disorders. British journal of haematology 2000;111:68-77.

40. Chatterjee S. Neutral sphingomyelinase: past, present and future. Chemistry and physics of lipids 1999;102:79-96.

41. Kempe DS, Akel A, Lang PA, et al. Suicidal erythrocyte death in sepsis. Journal of molecular medicine 2007;85:273-81.

42. Eichelbronner O, Sielenkamper A, Cepinskas G, Sibbald WJ, Chin-Yee IH. Endotoxin promotes adhesion of human erythrocytes to human vascular endothelial cells under conditions of flow. Critical care medicine 2000;28:1865-70.

43. Anniss AM, Sparrow RL. Variable adhesion of different red blood cell products to activated vascular endothelium under flow conditions. American journal of hematology 2007;82:439-45.

44. Vincent JL, Baron JF, Reinhart K, et al. Anemia and blood transfusion in critically ill patients. Jama 2002;288:1499-507.

45. Croce MA, Tolley EA, Claridge JA, Fabian TC. Transfusions result in pulmonary morbidity and death after a moderate degree of injury. The Journal of trauma 2005;59:19-23;

46. Netzer G, Hess JR. TRALI, transfusion, and acute lung injury: synergy in action? Critical care medicine 2010;38:981-2.

47. Netzer G, Shah CV, Iwashyna TJ, et al. Association of RBC transfusion with mortality in patients with acute lung injury. Chest 2007;132:1116-23.

48. Zilberberg MD, Carter C, Lefebvre P, et al. Red blood cell transfusions and the risk of acute respiratory distress syndrome among the critically ill: a cohort study. Critical care 2007;11:R63.

49. Gajic O, Rana R, Winters JL, et al. Transfusion-related acute lung injury in the critically ill: prospective nested case-control study. American journal of respiratory and critical care medicine 2007;176:886-91.

50. Gauvin F, Spinella PC, Lacroix J, et al. Association between length of storage of transfused red blood cells and multiple organ dysfunction syndrome in pediatric intensive care patients. Transfusion 2010;50:1902-13.

51. Kleinman S. A perspective on transfusion-related acute lung injury two years after the Canadian Consensus Conference. Transfusion 2006;46:1465-8.

52. Goldman M, Webert KE, Arnold DM, et al. Proceedings of a consensus conference: towards an understanding of TRALI. Transfusion medicine reviews 2005;19:2-31.

53. Toy P, Popovsky MA, Abraham E, et al. Transfusion-related acute lung injury: definition and review. Critical care medicine 2005;33:721-6.

54. Peters AL, van Hezel ME, Juffermans NP, Vlaar AP. Pathogenesis of non-antibody mediated transfusion-related acute lung injury from bench to bedside. Blood reviews 2015;29:51-61.

55. Vlaar AP, Juffermans NP. Transfusion-related acute lung injury: a clinical review. Lancet 2013;382:984-94.

56. Napolitano LM, Corwin HL. Efficacy of red blood cell transfusion in the critically ill. Critical care clinics 2004;20:255-68.

57. Vincent JL, Piagnerelli M. Transfusion in the intensive care unit. Critical care medicine 2006;34:S96-101.

58. Murphy GJ, Pike K, Rogers CA, et al. Liberal or restrictive transfusion after cardiac surgery. The New England journal of medicine 2015;372:997-1008.

59. Silliman CC, Voelkel NF, Allard JD, et al. Plasma and lipids from stored packed red blood cells cause acute lung injury in an animal model. The Journal of clinical investigation 1998;101:1458-67.

60. Mangalmurti NS, Xiong Z, Hulver M, et al. Loss of red cell chemokine scavenging promotes transfusion-related lung inflammation. Blood 2009;113:1158-66.

61. Silliman CC, Khan SY, Ball JB, Kelher MR, Marschner S. Mirasol Pathogen Reduction Technology treatment does not affect acute lung injury in a two-event in vivo model caused by stored blood components. Vox sanguinis 2010;98:525-30.

62. Silliman CC, Moore EE, Kelher MR, Khan SY, Gellar L, Elzi DJ. Identification of lipids that accumulate during the routine storage of prestorage leukoreduced red blood cells and cause acute lung injury. Transfusion 2011;51:2549-54.

63. Khan SY, Kelher MR, Heal JM, et al. Soluble CD40 ligand accumulates in stored blood components, primes neutrophils through CD40, and is a potential cofactor in the development of transfusionrelated acute lung injury. Blood 2006;108:2455-62.

64. Kelher MR, Masuno T, Moore EE, et al. Plasma from stored packed red blood cells and MHC class I antibodies causes acute lung injury in a 2-event in vivo rat model. Blood 2009;113:2079-87.

65. Vlaar AP, Hofstra JJ, Levi M, et al. Supernatant of aged erythrocytes causes lung inflammation and coagulopathy in a "twohit" in vivo syngeneic transfusion model. Anesthesiology 2010;113:92-103.

66. Tung JP, Fraser JF, Nataatmadja M, et al. Age of blood and recipient factors determine the severity of transfusion-related acute lung injury (TRALI). Critical care 2012;16:R19.

67. Patel SV, Kidane B, Klingel M, Parry N. Risks associated with red blood cell transfusion in the trauma population, a metaanalysis. Injury 2014;45:1522-33.

68. Vlaar AP, Binnekade JM, Prins D, et al. Risk factors and outcome of transfusion-related acute lung injury in the critically ill: a nested case-control study. Critical care medicine 2010;38:771-8.

69. Middelburg RA, Borkent-Raven BA, Janssen MP, et al. Storage time of blood products and transfusion-related acute lung injury. Transfusion 2012;52:658-67.

70. Toy P, Gajic O, Bacchetti P, et al. Transfusion-related acute lung injury: incidence and risk factors. Blood 2012;119:1757-67.

71. Lacroix J, Hebert PC, Fergusson DA, et al. Age of transfused blood in critically ill adults. The New England journal of medicine 2015;372:1410-8.

72. Steiner ME, Ness PM, Assmann SF, et al. Effects of red-cell storage duration on patients undergoing cardiac surgery. The New England journal of medicine 2015;372:1419-29.

73. Silliman CC, Bjornsen AJ, Wyman TH, et al. Plasma and lipids from stored platelets cause acute lung injury in an animal model. Transfusion 2003;43:633-40.

74. Silliman CC, Kelher MR, Khan SY, et al. Experimental prestorage filtration removes antibodies and decreases lipids in RBC supernatants mitigating TRALI in vivo. Blood 2014;123:3488-95.

75. Vlaar AP, Hofstra JJ, Kulik W, et al. Supernatant of stored platelets causes lung inflammation and coagulopathy in a novel in vivo transfusion model. Blood 2010;116:1360-8.

76. Wyman TH, Bjornsen AJ, Elzi DJ, et al. A two-insult in vitro model of PMN-mediated pulmonary endothelial damage: requirements for adherence and chemokine release. American journal of physiology Cell physiology 2002;283:C1592-603.

77. Blumberg N, Gettings KF, Turner C, Heal JM, Phipps RP. An association of soluble CD40 ligand (CD154) with adverse reactions to platelet transfusions. Transfusion 2006;46:1813-21.

78. Xie RF, Hu P, Li W, et al. The effect of platelet-derived microparticles in stored apheresis platelet concentrates on polymorphonuclear leucocyte respiratory burst. Vox sanguinis 2014;106:234-41.

79. Tuinman PR, Gerards MC, Jongsma G, Vlaar AP, Boon L, Juffermans NP. Lack of evidence of CD40 ligand involvement in transfusion-related acute lung injury. Clinical and experimental immunology 2011;165:278-84.

80. Rubin O, Delobel J, Prudent M, et al. Red blood cell-derived microparticles isolated from blood units initiate and propagate thrombin generation. Transfusion 2013;53:1744-54.

81. Gao Y, Lv L, Liu S, Ma G, Su Y. Elevated levels of thrombin-generating microparticles in stored red blood cells. Vox sanguinis 2013;105:11-7.

82. Jy W, Ricci M, Shariatmadar S, Gomez-Marin O, Horstman LH, Ahn YS. Microparticles in stored red blood cells as potential mediators of transfusion complications. Transfusion 2011;51:886-93.

83. Darbonne WC, Rice GC, Mohler MA, et al. Red blood cells are a sink for interleukin 8, a leukocyte chemotaxin. The Journal of clinical investigation 1991;88:1362-9.

84. Barshtein G, Manny N, Yedgar S. Circulatory risk in the transfusion of red blood cells with impaired flow properties induced by storage. Transfusion medicine reviews 2011;25:24-35.

85. Stanworth SJ, Grant-Casey J, Lowe D, et al. The use of fresh-frozen plasma in England: high levels of inappropriate use in adults and children. Transfusion 2011;51:62-70.