Blood Product Transfusions and Reactions

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KEYWORDS

• Blood products • Transfusion • Packed red blood cells • Fresh frozen plasma

KEY POINTS

- Blood product transfusions are an essential component of the practice of emergency medicine.
- From acute traumatic hemorrhage to chronic blood loss necessitating transfusion for symptomatic anemia, familiarity with individual blood products and their indications for transfusion is an essential tool for every emergency physician (EP).
- The advances made in transfusion medicine over the past few decades have ensured that administration of blood products has become safer than ever before, but significant risks still exist, and will continue to present EPs with diagnostic and treatment challenges.

Blood product transfusions are an essential component of the practice of emergency medicine. From acute traumatic hemorrhage to chronic blood loss necessitating transfusion for symptomatic anemia, familiarity with individual blood products and their indications for transfusion is an essential tool for every emergency physician (EP). Although the focus of this article is primarily on the transfusion of red blood cells, many of the concepts are applicable to the transfusion of all blood products, including platelets, cryoprecipitate, and (supernatant) fresh frozen plasma (FFP).

The history of blood transfusions dates back to the 1600s when British physician William Harvey first discovered the circulation of blood, followed closely by the first successful blood transfusion in 1665.[1] Over the past several decades, advances in blood transfusion medicine have made the practice of administering these transfusions safer and more accessible to the EP. According to the Red Cross, more than 30 million blood components are transfused per year in the United States.[2] EPs must be fully familiar with both the individual blood components and the potential reactions and complications of these transfusions.

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BLOOD PRODUCTS

Packed Red Blood Cells

Packed red blood cells (PRBCs) are used clinically to increase the hemoglobin and oxygen-carrying capacity in an anemic patient. PRBCs are procured from whole blood samples. From a single donation unit of whole blood, approximately 2 transfusable units of PRBCs are collected.[3] PRBCs are stored between 1° and 6°C in a solution containing citrate, phosphate, dextrose, and adenine as well as nutrient additives, which confers a shelf life of approximately 42 days for each unit.[3] One unit of PRBCs given to an average adult will elevate the hemoglobin by about 1 g/dL and the hematocrit by about 3% (**Table 1**).[7] Transfusion guidelines for PRBCs are controversial, and recommendations vary between professional societies. In 2012 the American Association of Blood Banks (AABB) released a clinical practice guideline for transfusion of PRBCs based on a systematic review of multiple randomized clinical trials. The AABB recommends that in hospitalized, stable patients a threshold of 7 to 8 g/dL should be used to guide transfusion based on high-quality evidence.[4] For individuals with preexisting cardiovascular disease, the threshold should be 8 g/dL or less, although this is a weak recommendation by the AABB based on moderate-quality evidence.[4] Ultimately, transfusion decisions should be based on the clinical presentation of the patient in conjunction with the clinical gestalt of the physician.

Platelets

Platelets can be isolated for transfusion either from whole blood donations, which often require multiple or pooled donors to produce a unit, or from platelet apheresis procedures, in which a single donor can provide sufficient platelets for a transfusion unit.[8] Platelets are stored at 22°C, which does increase the risk for bacterial contamination in comparison with PRBCs, which are stored at much lower temperatures.[8] Platelets can be stored safely for 7 days.[8] Platelet transfusions tend to be dosed as a "6-pack" of platelets, which contains 6 units of platelet concentrate from multiple donors or a single apheresis unit, and can be expected to raise the platelet count by 40,000 to 60,000/μL (see **Table 1**).[3] Although there is no definitive trigger for platelet transfusion, current data support the transfusion of platelets for counts less than or equal to 10,000.[8]

Fresh frozen plasma

FFP can also be prepared from whole blood or be collected by apheresis, and contains normal levels of stable clotting factors, albumin, and immunoglobulins.[5] One unit of FFP is usually about 200 to 250 mL in volume.[3] It is stored frozen at −18° to −30°C and then thawed between 30° and 37°C in a water bath under continuous agitation.[5] After thawing, the FFP should be administered as soon as possible,

Table 1
Recommendations for dosing of blood products

Packed red blood cells	10 mL/kg (children) 1 unit per 1 g/dL increase desired (adults)
Platelets	5–10 mL/kg (children) 1 "6-pack" or 1 aphresis unit per 40–60,000/μL increase (adults)
Fresh frozen plasma	10–15 mL FFP/kg (all patients)
Cryoprecipitate	1 cryo unit per 5 kg for 100 mg/dL fibrinogen increase (all patients)

Data from Refs.[3–6]

but no later than 24 hours after thawing.[5] Once again, no strict guidelines exist for transfusion thresholds; however, when the prothrombin time or activated partial thromboplastin time is more than 1.5 times the upper limit of normal, the accepted practice is to transfuse in the appropriate clinical circumstances (see **Table 1**).[5]

Cryoprecipitate

When 1 unit of FFP is thawed to 1° to 6°C, centrifuged for 6 minutes, and the supernatant removed, the remaining insoluble precipitate along with about 5 to 15 mL of plasma constitutes cryoprecipitate.[6] The cryoprecipitate is then refrozen and stored at −18°C or lower, and can be used for up to 12 months after the original preparation of product.[6] Cryoprecipitate contains a higher concentration of factor VIII, von Willebrand factor, and fibrinogen, and 1 unit of cryoprecipitate will increase the fibrinogen level by 5 to 10 mg/dL in the average adult (see **Table 1**).[6] Similar to the other blood components, there are no definitive transfusion guidelines for cryoprecipitate; however, general recommendations suggest its use in patients with fibrinogen levels of less than 1.0 g/L in the setting of severe bleeding or disseminated intravascular coagulation.[6]

BLOOD PRODUCT TYPING AND ANTIBODIES

Perhaps the most essential aspect of a blood product transfusion is matching the donor and recipient blood types as accurately as possible to avoid an adverse reaction to the transfusion. There are 3 major requirements that must be satisfied with any transfusion. The first is the matching of the ABO groups; second, the Rh(D) groups must be matched for premenopausal female recipients; finally, there must be a screening for any antibodies in the recipient sample that may cross-react with the donor sample antigens.[9] Rh(D) matching in Rh(D)-negative premenopausal female patients is important in preventing sensitization and the development of immunoglobulin (Ig)G anti-Rh(D) antibodies, which may lead to hemolytic disease of the newborn in future pregnancies and affect the viability of the pregnancy. The type and screen that is performed before a transfusion will not detect rare nonpolymorphic antigens; fortunately, however, these antigens are rarely of clinical significance during a transfusion.[9]

Although the major blood group antigens of A, B, and O are probably the most significant antigens to match for, there is a myriad of antigens that must be cross-matched before a transfusion to avoid the morbidity and mortality associated with mismatched blood products. The A, B, and the lack of A or B antigens, which is designated the O blood type, are responsible for most of the immediate severe transfusion reactions. The Rh blood group system additionally contains more than 45 independent antigens that can complicate transfusions in patients who have been previous recipients of blood products.[10] The most common Rh antigens are D, C, and E, but there are multiple glycoproteins in the system that can be immunoactive and lead to transfusion reactions.[10]

O Rh(D)-negative blood is considered the universal donor, and is typically the stocked blood in the emergency department (ED) setting that is readily available for rapid transfusion when needed. A review of the Retrovirus Epidemiology Donor Study from the United States revealed that the highest percentage of Group O is found in Hispanic, North American Indian, and non-Hispanic black donors, while the Rh(D) phenotype is found most commonly in non-Hispanic white donors and North American Indian donors.[11] Although generalizations about the ethnic predispositions to certain blood types may be of use in recruiting donors, extensive cross-matching of blood for transfusion is the most essential component of transfusion medicine.

ADVERSE TRANSFUSION REACTIONS

Febrile Nonhemolytic Transfusion Reactions

Febrile nonhemolytic transfusion reactions (FNHTRs) are the most commonly reported adverse reactions to transfusion, and occur usually within the first 2 hours of initiation of transfusion (**Table 2**).[12,13] Although FNHTRs are benign by nature, the signs and symptoms associated with them are similar to those of more ominous transfusion reactions. Therefore, with FNHTRs the transfusion must be stopped and a thorough investigation initiated to rule out potential fatal adverse reactions (**Fig. 1**). By strict definition an FNHTR is the increase in temperature of at least 1°C from baseline, accompanied often by chills, rigors, and discomfort, although it has been reported that symptoms such as chills and discomfort can occur in the absence of temperature change.[12] These reactions are caused by 2 different mechanisms, the first of which is the transfusion of leukocyte antigens that result in the activation of the cytokine cascade in the recipient.[12,14] The second is the transfusion of cytokines from the stored blood products that have the ability to cause an inflammatory immune response in the recipient.[12,14] Platelet transfusion carries a higher risk of FNHTRs than PRBC transfusion because of the higher concentration of both leukocyte antigens and cytokines in the platelet preparations.[12] Febrile reactions to platelet transfusions have been reported with a rate ranging from 1% to 38%, whereas for red cell transfusions the rate is generally 0.3% to 6%.[13] Premedication with acetaminophen and diphenhydramine is a common practice to prevent these FNHTRs. However, the literature is controversial as to whether this practice of premedication is of benefit to patients. A study by Wang and colleagues[15] in 2002 found that premedication does not significantly reduce the incidence of FNHTRs in platelet transfusions. By contrast, in 2008 Kennedy and colleagues[13] found that the administration of these premedications may suppress febrile reactions, especially in the subset of patients who receive multiple transfusions; however, it did not decrease the overall rate of transfusion reactions in the subset of patients studied. Premedication comes with its own inherent risks, including the masking of the early signs of a potential fatal transfusion reaction. As to date there has been no definitive opinion offered about the use of premedication before transfusion, the decision to use premedication should be deferred to the individual physician and may be informed by local practice patterns.

Allergic Transfusion Reactions

Allergic transfusion reactions (ATRs) are also a common occurrence, with a frequency of 1% to 3% of all transfusions (see **Table 2**).[16,17] ATRs are thought to be due to a type I hypersensitivity reaction, with IgE antibodies reacting with antigens

Table 2
Estimated incidence of significant transfusion-related reactions (independent of mortality rates)

Acute hemolytic transfusion reaction	1:76,000
Febrile nonhemolytic transfusion reaction	0.1%–1.0%
Allergic (urticarial) transfusion reaction	1%–3%
Anaphylactic/anaphylactoid transfusion reaction	1:20,000–50,000
Transfusion-related acute lung injury	1:10,000

Data from Weinstein R. 2012 clinical practice guide on red blood cell transfusion. Washington, DC: American Society of Hematology; 2014. Available at: http://www.hematology.org/Practice/Guidelines/9138.aspx.

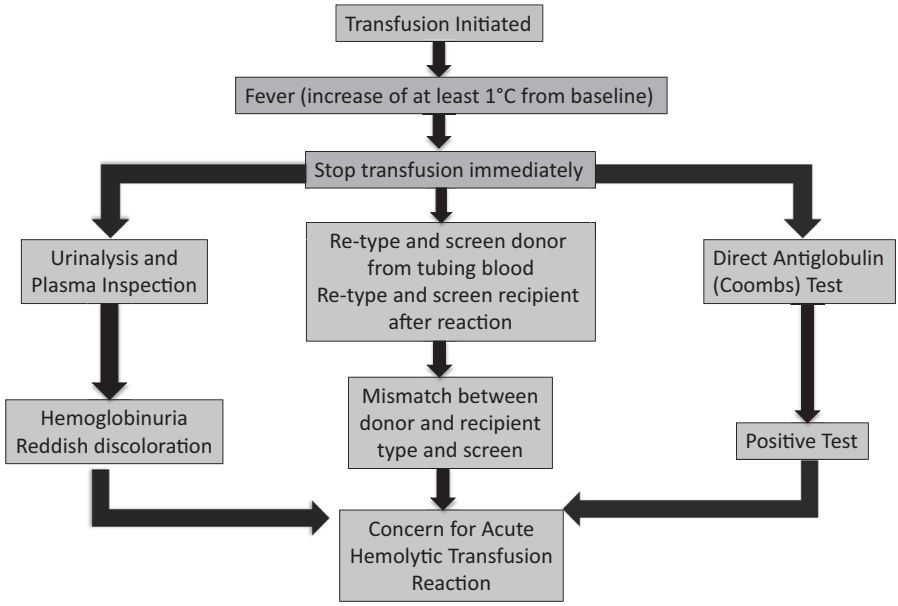

Fig. 1. Basic laboratory workup for possible acute hemolytic transfusion reaction after fever in a patient receiving blood product transfusion.

to activate mast cells and basophils and cause resultant histamine release.[14,16] Simple ATRs tend to present within the first 2 hours after initiation of transfusion with urticarial rashes and pruritus, although patients can experience anaphylactic reactions to transfusions as well, which may present with symptoms including bronchospasm, respiratory distress, angioedema, or hypotension.[13] Anaphylactic reactions occur more commonly in patients with IgA deficiency who have circulating IgG anti-IgA antibodies when they are given whole blood transfusions that are not IgA-poor or washed, but they can potentially occur in any patient.[14] In patients with a history of severe allergic or anaphylactic reactions who require repeat transfusion, saline-washed red cells should be used.[14] A common practice is to administer diphenhydramine before transfusion to prevent allergic reaction, although there have been no studies showing a significant difference in the incidence of allergic transfusion reactions secondary to premedication.[18]

Hemolytic Transfusion Reactions

Hemolytic transfusion reactions (HTRs) are a potentially fatal complication of blood product transfusions. Fatalities from HTR, though still a rare event, do continue to occur in approximately 1 in 1.5 million to 1 in 1.8 million transfusions.[19] HTRs can be generally classified as either acute or delayed in presentation. The acute form results from the immune-mediated destruction of incompatible donor red cells by preexisting recipient antibodies, and can occur either intravascularly or extravascularly.[20] Acute reactions occur within 24 hours of the transfusion.[20] Delayed reactions occur approximately 7 to 10 days after the initial transfusion of seemingly compatible blood, with the destruction of donor red cells by the immune response of the recipient to a donor antigen to which the recipient was previously exposed.[20] Although most HTR-related deaths are secondary to acute hemolysis, there does exist a smaller subset of deaths from delayed hemolysis, although this is a much rarer occurrence.[20]

An HTR presents with chills or rigors, fever, back or flank pain, hypotension, renal failure, and disseminated intravascular coagulation primarily.[14,21] The severity of an HTR depends on the recipient antibody, which is most severe in ABO mismatch, and the volume of blood transfused, which usually requires the transfusion of more than 200 mL to result in fatality.[20] Although improvements in blood banking and safety have decreased the number of fatalities secondary to ABO mismatch, there has been an increase over recent years in fatalities from non-ABO antibodies. Between the years of 2005 to 2007, 69.2% of all fatal HTRs were thought to have been caused by non-ABO antibodies.[20] Given that many of these severe HTRs are difficult to treat even with aggressive supportive care, the most effective strategy to decrease fatal HTRs focuses on prevention of events, with multiple checks and rechecks of both donor and recipient sample to ensure compatibility.

TRANSFUSION-ASSOCIATED CIRCULATORY OVERLOAD AND TRANSFUSION-RELATED ACUTE LUNG INJURY

Transfusion-associated circulatory overload (TACO) and transfusion-related acute lung injury (TRALI) are 2 of the more severe and lethal adverse reactions to the transfusion of blood and blood products (see **Table 2**). These entities can cause significant respiratory distress, at times requiring advanced airway management and aggressive pulmonary support. These disorders may present very similarly after transfusion and may even coexist within the same patient; however, the identification of the primary respiratory issue, be it TACO or TRALI, is essential in the management of these patients.

TACO is a relatively common entity that may be underdiagnosed in transfusion populations. The incidence of TACO in previous studies has been estimated to be as low as less than 1% and as high as 11% in certain populations such as the elderly.[22] TACO results from the transfusion of blood products into a patient with impaired cardiac reserve, resulting in hydrostatic cardiogenic pulmonary edema and respiratory distress.[23] Patients at the extremes of age and those receiving massive transfusions are at highest risk of TACO.[24] The diagnosis of TACO is made in a patient without significant existing pulmonary edema before transfusion who develops dyspnea and hypoxemia with a partial pressure of oxygen/fraction of inspired oxygen (Pao_2/Fio_2) ratio of 300 mm Hg or less, or oxygen saturation (Spo_2) of 90% or less within 6 hours of transfusion, in the setting of bilateral infiltrates on chest radiograph with clinically evident left atrial hypertension.[23] In previous studies and reviews, mortality in patients with TACO was found to be between 5% and 20%.[22,23] Treatment of TACO is centered on expedient volume reduction with diuresis and noninvasive or invasive ventilation as needed, much as an EP would treat an exacerbation of acute congestive heart failure.[22]

TRALI is one of the most common causes of transfusion-associated morbidity and death. TRALI presents similarly to TACO, with dyspnea and hypoxemia that typically occurs in the first 6 hours after transfusion, although symptoms usually start within 1 to 2 hours and can present as late as 48 hours after transfusion.[24] The pathophysiology of TRALI has been proposed to be increased vascular permeability secondary to donor antileukocyte antibodies, and biologically active substances with neutrophil-priming activity that lead to the development of noncardiogenic pulmonary edema.[25,26] TRALI has been formally defined by a working group at the National Heart, Lung and Blood Institute as new acute lung injury with onset of symptoms or signs within 6 hours of the initiation of transfusion or the end of the transfusion with no other reason for acute lung injury beyond the transfusion itself.[25] Acute lung injury is defined as an acute-onset

process with bilateral infiltrates on chest radiograph and hypoxemia with a Pa_{O_2}/Fi_{O_2} ratio 300 mm Hg or less or Sp_{O_2} 90% or less on room air, yet with pulmonary artery occlusion pressure of 18 mm Hg or less or a lack of clinical evidence of left atrial hypertension.[25,27] The management of TRALI primarily focuses on respiratory support with mechanical intervention as needed, along with other supportive agents such as vasopressors for sustained hypotension.[24] Corticosteroid efficacy has thus far remained unproven in the literature, and the use of diuretics in these patients is controversial.[24] Because TRALI is not secondary to fluid overload and instead involves microvascular injury, diuretics may be detrimental to the patient, and they may respond to fluid administration.[24] Fortunately, most cases of TRALI resolve within 96 hours.[20]

Distinguishing between these two entities can be difficult, especially in those patients who may have elements of both disease processes present; furthermore, there have been no laboratory studies or other tests reliably able to distinguish between the two. For the EP, the chest radiograph often does not distinguish significantly between the two conditions.[22] Signs of cardiomegaly on chest radiograph may suggest the presence of underlying cardiac dysfunction that may predispose these patients to TACO; however, the same patient with underlying cardiomegaly may also develop TRALI, making diagnosis based on chest radiography difficult. Echocardiography has also been used to distinguish between the two conditions, although its usefulness has not been determined in the literature.[22] B-Type natriuretic peptide (BNP) is often used as a measurement of ventricular strain and volume overload, although in the setting of pulmonary edema after transfusion the BNP may not increase at a rate significant enough to be useful in differentiating between TACO and TRALI. BNP has found to be elevated in patients without left ventricular dysfunction in the setting of acute lung injury, which further complicates its utility in diagnosis.[22] However, a study by Zhou and colleagues[28] investigated BNP levels before and after transfusion, and found that an increase in BNP of at least 50% after transfusion had 81% sensitivity and 89% specificity for the diagnosis of TACO. Although drawing BNP on every patient receiving a transfusion is certainly not cost-effective, there may be some utility to drawing a pretransfusion BNP in particularly high-risk patients, but this has not been further studied in the literature. Invasive testing such as pulmonary edema fluid protein concentration measurements may be of use in the final determination between the two entities; however, this is not clinically useful to the EP. Ultimately, in the emergency care setting the patient should be treated supportively in both situations, with specific diuretic therapy reserved for those patients with clinical signs of volume overload such as elevated jugular venous distention in an appropriate patient setting (**Fig. 2**).

TRANSFUSION-ASSOCIATED SEPSIS AND INFECTION TRANSMISSION

Transfusion-associated sepsis (TAS) is another significant adverse event in the setting of blood transfusion that carries significant morbidity and mortality, accounting for 17% to 22% of all transfusion-related fatalities.[30] According to multiple studies including the Serious Hazards of Transmission (SHOT), the French Haemovigilance Study, and the Food and Drug Administration fatality reports, the incidence of clinically apparent bacterial sepsis is much higher than that of transfusion-transmitted viral infections, which is attributed to the increased screening of donor products for viral contamination.[30] Although platelets have typically been identified as the main culprit in TAS, given their storage at room temperature that provides an ideal environment for bacterial replication, TAS and viral infection transmission can occur with any blood product.[30] The most common bacteria in packed red cells is *Yersinia enterocolitica*,

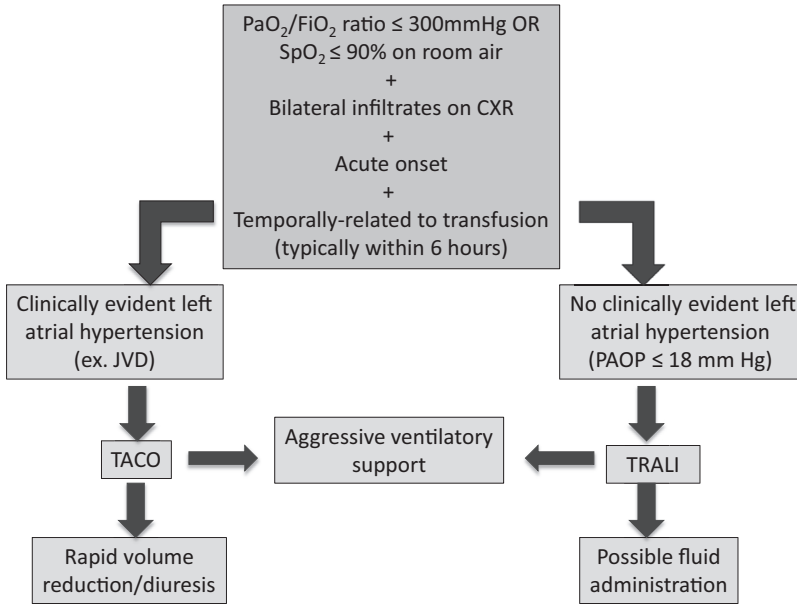

Fig. 2. Diagnostic and treatment similarities and differences between transfusion-related circulatory overload (TACO) and transfusion-related acute lung injury (TRALI). CXR, chest radiograph; FiO_2, fraction of inspired oxygen; JVD, jugular venous distention; PaO_2, partial pressure of arterial oxygen; PAOP, pulmonary artery occlusion pressure; SpO_2, oxygen saturation.

which grows well in the citrate-rich and iron-rich environment, but *Serratia* species and *Pseudomonas* species can also be present in donor samples.[14,31] Platelets tend to breed skin flora, most likely *Staphylococcus aureus, Escherichia coli, Bacillus* species, *Salmonella* species, *Streptococcus* species, and *Klebsiella* species, among others.[14,30] In the setting of platelet transfusion, fatalities are most often associated with *Klebsiella* species (17.3%), and of all TAS fatalities secondary to platelet transfusion, gram-negative organisms are the culprit in 60% of fatalities, likely secondary to the endotoxin component of many of these organisms, with gram-positives accounting for the other 40%.[30,32] Viral transmission rates have dropped dramatically over the last 20 to 30 years with the advent of intensive screening processes, with transmission rates of hepatitis B and C and human immunodeficiency virus (HIV) at very low levels (**Table 3**).[31] TAS will manifest with fevers, rigors, vomiting, and hypotension, which usually start within 2 hours of the transfusion, and may progress to septic shock.[30,32]

Table 3
Risks of infection per unit transfused of common viruses, from 2001 data

Hepatitis B	1:180,000
Hepatitis C	1:1,600,000
HIV	1:1,900,000

Abbreviation: HIV, human immunodeficiency virus.

Data from Hillyer CD, Josephson CD, Blajchman MA, et al. Bacterial contamination of blood components: risks, strategies and regulation: joint ASH and AABB educational session in transfusion medicine. Hematology Am Soc Hematol Educ Program 2003:575–89.

EPs must be vigilant in considering TAS as a possible diagnosis, as many of these febrile reactions after transfusion are often considered to be FNHTRs rather than the early signs of TAS.[32] Blood products are routinely screened for hepatitis B virus, HIV, hepatitis C virus, *Treponema pallidum*, human T-cell lymphotropic virus, West Nile virus, cytomegalovirus, and *Trypanosoma cruzi*, and all platelets are additionally screened for bacterial contamination.[20,32] However, there are multiple bacterial and viral species that can potentially cause disease in the recipient but are not routinely screened for in donor samples, including hepatitis A virus, parvovirus B19, dengue fever virus, *Babesia* species, *Plasmodium* species, *Leishmania* species, *Brucella* species, and variant Creutzfeldt-Jakob disease prions.[20] Screening has dramatically reduced the numbers of blood product-borne infections in the United States and worldwide; in addition, most of the platelets currently being transfused in the United States are single-donor platelets, which also decreases the risk of bacterial and viral contamination via source control.[20] However, TAS is still an ever-present entity that needs to be on the differential diagnosis of any practicing EP, and antibiotic coverage should be tailored to ensure activity against those pathogens most commonly found in specific blood products. For PRBCs, appropriate antibiotics may include aminoglycosides, trimethoprim-sulfamethoxazole, or β-lactams to cover for bacteria such as *Yersinia*, *Serratia*, and *Pseudomonas*. For platelet-associated TAS, third- or fourth-generation cephalosporins, trimethoprim-sulfamethoxazole, aminoglycosides, or fluoroquinolones may be used to cover bacteria such as *Staphylococcus, E coli, Bacillus, Salmonella*, and *Streptococcus*.

TRANSFUSION-ASSOCIATED GRAFT-VERSUS-HOST DISEASE

Transfusion-associated graft-versus-host disease (TA-GvHD) is an extremely rare but almost uniformly fatal event occurring after transfusion. When immunocompetent lymphocytes are transfused into an immunocompromised recipient, the donor lymphocytes mount an immunologic response against the recipient (host) cells.[33] Onset of TA-GvHD occurs within 8 to 10 days of transfusion, with death occurring within 1 to 3 weeks after symptoms.[14] Symptoms of TA-GvHD include maculopapular rash, fever, elevated liver enzymes, hepatomegaly, jaundice, and gastrointestinal symptoms.[14,33] Prevention of this entity is the hallmark of "treatment," as no treatment modalities have ever been shown to treat TA-GvHD and the mortality rate is nearly 100%.[33] Although treatment options may be futile, this is a rare but important diagnosis to recognize in the ED in an immunocompromised patient with recent transfusion who may be presenting with these symptoms. In addition, EPs can play a role in the prevention of this disease by identifying patients at risk for TA-GvHD and ensuring that any transfusions they receive are irradiated to eliminate donor lymphocytes. Populations that are particularly at risk include patients with congenital immunodeficiency syndromes, patients who have received bone marrow transplants, and patients with Hodgkin disease.[34]

TRANSFUSION CONSIDERATIONS: CULTURAL AND RELIGIOUS CONSIDERATIONS

Cultural or religious beliefs are an important consideration, and in some cases can complicate decision making regarding administration of blood products. This dilemma can pose a particular challenge when the patient is critically ill and in need of emergency blood products.

Jehovah's Witnesses have approximately 1 million active members in North America and 6 million worldwide, and practicing Jehovah's Witnesses may not accept any transfusions of whole blood or any of the individual components of blood.[35] In this

subset of patients, alternative bloodless options often must be explored to optimize medical care. Although legal precedent does support the ability of adult Jehovah's Witnesses to refuse blood products and make their own treatment decisions, an area of controversy may arise when the child of a Jehovah's Witness is critically ill and in need of blood products. Minors are not capable of providing informed consent, and if the decisions made by the parents place the patient at high risk for morbidity and mortality, it is the duty of the EP to intervene, including seeking assistance from the ethics committee or legal counsel to make the best decision for the patient.[35] In a life-threatening situation where blood is required for emergent stabilization, which is particularly true in acute traumatic situations, the transfusion of a minor should be considered regardless of the presence of a court order.[35]

SPECIAL SITUATIONS: MASSIVE TRANSFUSION

One of the primary indications for the use of transfusions in the ED is the management of acute hemorrhagic shock in trauma. These patients often require massive transfusions, generally defined as the anticipated need for greater than 10 units of PRBCs in 24 hours.[36] The bulk of the research on massive transfusions and massive transfusion protocols has come from the military literature; however, these concepts have become common practice in civilian EDs. Massive transfusions of isolated red blood cells can quickly lead to severe coagulopathies, which are often exacerbated by hypothermia, acidosis, and other derangements that are common in the setting of trauma.[29,36] A widely used practice is the transfusion of a 1:1:1 ratio of PRBCs to platelets to FFP. Although the goal is to transfuse a product that is as close to whole blood as possible, the 1:1:1 ratio transfusion provides a hematocrit of 29%, a platelet count of 85,000/μL, and approximately 60% of normal clotting activity, which differs from whole blood.[36] The initiation of massive transfusion protocols typically involves O-negative blood; however, rapid typing and cross-matching of blood should be performed to reduce the risk of transfusion-related complications during a massive transfusion and to minimize the amount of O-negative blood used.[37]

Although massive transfusions carry the risks of any transfusion, particular electrolyte and volume abnormalities are more commonly associated with massive transfusions. Careful monitoring of coagulation factors, blood counts, and electrolytes must occur during massive transfusions. Common electrolyte abnormalities include hypocalcemia from the citrate used in the storage of PRBCs, and hyperkalemia secondary to the increase in potassium concentration of stored units of PRBCs.[36,37] Acute lung injury is also commonly associated with massive transfusions, and recent data suggest that there may be a causal relationship between massive transfusions and acute lung injury.[38] These massive transfusion ratios of 1:1:1 have been shown in both military and civilian data to greatly improve morbidity and mortality, and should be strongly considered in the appropriate setting.[36,37,39]

SUMMARY

Blood product transfusions are common practice in emergency medicine, and adverse reactions, though rare, are often associated with significant morbidity and mortality. Vigilance and recognition of these entities, especially early in the course of disease, can often make a significant impact on the disease course and, in the case of the most severe transfusion-related complications, the survivability of these patients. The advances made in transfusion medicine over the past few decades have ensured that administration of blood products has become safer than ever

before, but significant risks still exist and will continue to present EPs with diagnostic and treatment challenges.

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