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Review Article

TRANSFUSION-RELATED COMPLICATIONS IN INTENSIVE CARE UNIT (ICU), RISK FACTORS, PREVENTION AND MANAGEMENT: A LITERATURE REVIEW

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ABSTRACT

Background: *Blood transfusion is a cornerstone of supportive therapy in intensive care units (ICUs), particularly among critically ill patients with anemia, trauma, sepsis, and massive hemorrhage. Despite strict blood safety protocols, transfusion-related complications remain a significant cause of morbidity and mortality in ICU settings.* ***Objective:*** *To comprehensively review transfusion-related complications in ICU patients, identify major risk factors, and summarize current strategies for prevention and management.* ***Methods:*** *A narrative literature review was conducted using PubMed, Embase, Web of Science, and Google Scholar. Original studies, systematic reviews, and review articles addressing transfusion-related complications in adult ICU populations were included. Data were synthesized to categorize complications, associated risk factors, pathophysiological mechanisms, and evidence-based management approaches.* ***Results:*** *Transfusion-related complications in the ICU include immunologic reactions such as acute and delayed hemolytic transfusion reactions, febrile non-hemolytic reactions, transfusion-related acute lung injury (TRALI), transfusion-associated graft-versus-host disease, post-transfusion purpura, and allergic or anaphylactic reactions. Non-immunologic complications include transfusion-associated circulatory overload, transfusion-transmitted infections, massive transfusion–related metabolic disturbances, and iron overload. Advanced age, female sex, comorbid cardiac or renal disease, positive fluid balance, massive or repeated transfusions, shock, smoking, and chronic alcohol use were consistently identified as key risk factors. Early recognition, immediate cessation of transfusion, supportive care, and preventive strategies such as leukoreduction, irradiation, restrictive transfusion strategies, and adherence to massive transfusion protocols significantly reduce adverse outcomes.* ***Conclusion:*** *Transfusion-related complications remain common and potentially life-threatening in critically ill patients. Improved awareness, early diagnosis, risk stratification, and implementation of preventive transfusion strategies are essential to enhance patient safety and optimize outcomes in ICU practice.*

Keywords: Blood Transfusion; Intensive Care Units; Massive Transfusion; TRALI

INTRODUCTION

A blood transfusion is a typical therapeutic intervention for many hospitalized patients. Numerous indications for transfusion, including anemia and coagulopathy with deficiencies in single or multiple coagulation components, such as platelets or coagulation factors. Although it can be a life-saving therapy, it can also lead to serious adverse effects, and physicians must be aware of the pathophysiology, initial management, and risks of each type of transfusion reaction. Early detection, interruption of the transfusion, and resuscitation are all required for the initial therapy of blood transfusion reactions.

Historical Review of Blood Transfusion Medicine

In 1628, William Harvey, an English physician, discovered the circulation of blood (1). Shortly afterwards, the first reported successful blood transfusion occurred in England in 1665, where canines were kept alive by transfusing blood from other dogs (1).

The first human blood transfusion was performed in 1795 in Philadelphia, although he did not publish this information (1,2). In 1818, the first successful human blood transfusion to treat postpartum hemorrhage, using the patient's spouse as a donor, occurred (1,2). Moreschi describes the antiglobulin reaction. The antiglobulin test is a direct method for visualising an antigen-antibody reaction that has happened (1,2).

The first hospital blood bank in the United States was founded by Bernard Fantus in Chicago in 1937. A variety of blood components, including packed red blood cell (PRBC) concentrate, platelet concentrate, fresh frozen plasma, and cryoprecipitate, were discovered in 1960 (3).

Critically ill patients: Blood transfusion

Blood transfusions are a vital component of life support; they have become an essential aspect of modern medical treatment, particularly to allow ever-increasing aggressive therapy for patients who are older, sicker, and disabled (4).

During a hospital stay, between 30 and 50 per cent of intensive care unit patients receive a red blood cell transfusion. In the intensive care unit, approximately 60% of patients have hemoglobin levels below 12 g/dL, and approximately 97% are anemic, with hemoglobin levels below 12 g/dL for women and 14 g/dL for men by the 8th day in the ICU (4,5). Anemia in the ICU is commonly multifactorial, with the most common etiology being anemia of chronic disease (5).

Critically ill patients' transfusion requirements are due to many factors, including anemia in critically ill patients due to chronic anemia that already existed, acquired anemia, acute trauma, hemodilution, surgical procedures, bleeding in the gastrointestinal tract, frequent blood sampling, decreased erythropoietin synthesis, iron deficiency, increased hemolysis, and nutritional deficiencies (5). Life-threatening multiple organ failure as a consequence of sepsis induced by a dysregulated host response to infection is common among critically ill patients, and it is frequently accompanied by reduced oxygen delivery and organ hypoperfusion (6).

Transfusion in traumatic patients is a life-saving procedure because excessive bleeding leads to coagulopathy due to increased fibrinolysis, hemodilution, acidosis, hypothermia, and clotting factor deficiency. Transfusion replenishes blood volume lost, preserving tissue oxygenation and restoring coagulation factors (7,8).

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In severe burns, the pathophysiology of postburn anemia includes direct erythrocyte destruction, decreased erythropoietin responsiveness in the marrow, and repeated phlebotomy (9,10).

REVIEW OF LITERATURE

The current review was a literature review aimed at summarising the existing literature. To find relevant studies that investigated transfusion-related Complications, we searched various databases. The key databases searched for this literature review included PubMed, Web of Science, and Embase. Furthermore, the reference section of the potential studies and Google Scholar were also explored to enrich the literature review. The literature review included original studies, review articles, and systematic reviews. All studies that discussed transfusion-related complications in the ICU were included.

Transfusion-Related Complications

Even with blood safety protocols, transfusion-related adverse responses continue to be a significant contributor to transfusion recipient morbidity and mortality (11,12).

Critically ill patients are more likely to experience transfusion-related side effects due to increased exposure to transfusions and susceptibility to pulmonary complications. Transfusion responses range in intensity from trivial to life-threatening and can occur during the transfusion (immediate reactions) or within days to weeks after the transfusion (delayed reactions). They can be either immunologic or non-immunologic (13).

Table 1: Risk Factors for the occurrence of Blood Transfusion reactions

Study and year	Design	Risk factors
Muche Y et al. (2023). (14)	Observational	The older age group above 46 years was more at risk for transfusion reactions.
		Female sex
		A significant association between a history of blood transfusions and transfusion reactions.
		A significant association between a history of abortions and transfusion reactions.
		A correlation between older blood transfusions (more than 20 days old) and a higher risk of transfusion reactions.
		Number of units transfused: A higher likelihood of transfusion reactions with multiple units of blood transfused.
Van den Akker TA et al. (2022). (15)	Review	Heart failure, renal failure, extremes of age
Hu L et al. (2021). (16)	Systemic Review	Older adults may respond differently to transfusions than younger individuals.
		Sex-based differences in transfusion practices and outcomes.
		The amount of blood transfused, notably higher transfusion volumes, potentially increases the risk.
		Tobacco use & alcohol use potentially interact with transfusion-related complications.
		Patients in shock have more severe underlying conditions and may respond differently to transfusion compared to those not in shock.
De Cloedt L et al. (2019). (17)	Review	Age extremes and Sex differences were high risk for transfusion reactions.
		Positive fluid balance before transfusion increased the risk of TACO.
		The number and type of products transfused were significantly related to transfusion reactions.
		Cardiovascular and renal comorbidities.
Kuldanek SA et al. (2019). (18)	Review	Older age group were at high risk for transfusion reactions,
		Tobacco use & chronic alcohol abuse.
		Positive fluid balance
		Hematologic malignancy had a significant effect on the occurrence of transfusion-related complications.
Roubinian N et al. (2018). (19)	Review	Chronic alcohol abuse & tobacco use.
		The number of units transfused was associated with the occurrence of transfusion reactions.
		Shock before transfusion was significantly associated with increased transfusion reactions.
		Positive fluid balance before transfusion.
		Cardiovascular and renal comorbidities
Menis M et al. (2014). (20)	Retrospective Cohort	Age, sex & race.
		Tobacco use.
		The more transfusion units, the greater the risk.
		Pulmonary fibrosis.
Pearl Toy et al. (2012). (21)	Case -Control study	Sex difference.
		Chronic alcohol abuse and smoking.
		Positive fluid balance.
		Shock before transfusion.

Immunologic transfusion reactions

Allogenic transfused blood cells and plasma proteins are foreign substances that induce an immune response in the recipient, and plasma contains antibodies and other immune mediators that can react with recipient cells, leading to an immunologic reaction (22). Hemolytic reactions occur due to red cell incompatibility, while febrile responses occur due to leucocyte and platelet antigens (23).

Pathophysiology of Immunologic Transfusion Reactions

Acute hemolytic transfusion reactions (AHTRs) are the leading cause of transfusion-related mortality, resulting from recipient plasma

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antibodies reacting with donor red blood cell antigens, and the most common incompatibility is the blood group system ABO. Nevertheless, there may also be additional responses to other "less significant" antigens, such as Duffy and Kell (22). The prevalence of acute hemolytic transfusion reactions (AHTRs) has been estimated at approximately 1 in 70,000 per blood product transfused (23).

Febrile non-hemolytic Transfusion reactions (FNHTR)

Febrile responses can occur in the absence of hemolysis, with antibodies targeting human leukocyte antigen (HLA) on red blood cells as one possible cause. Commonly observed in individuals who have undergone numerous transfusions or multiparas. Another potential factor is the cytokines released by white blood cells during storage (24).

Among the risk factors are transfusions of more than 6 units of leukocyte-depleted packed red blood cells and primary hematologic disorders (15,16).

Transfusion Related Acute Lung Injury (TRALI)

Acute lung injury is the second most frequent cause of transfusion-related mortality (25). TRALI results from patient and blood component factors that activate neutrophils and damage endothelial cells, leading to capillary leak and a "perfect inflammatory storm" that causes significant lung injury (26). Mild to moderate TRALI is frequently overlooked. Non-cardiogenic pulmonary edema is the result of TRALI, which is characterised by a sudden onset of respiratory discomfort within six hours post-transfusion (26,27).

It may be categorised as TRALI type I in individuals without additional risk factors for ARDS. In contrast, those with risk factors or a history of moderate ARDS may be classified as TRALI type II, provided their respiratory status remains stable for 12 hours before the transfusion (25). The risk factors can be classified as recipient-related and blood component-related. Thrombotic microangiopathy, hematologic cancers, massive transfusion, current smoking, chronic alcohol misuse, positive fluid balance, end-stage liver disease, sepsis, surgical interventions for liver transplantation, and noncardiogenic shock are all linked to an increased risk of TRALI (16,17).

Delayed hemolytic transfusion reactions

The patient has developed sensitivity to a red blood cell antigen from a previous transfusion, with very low antibody levels and negative pretransfusion tests. In the liver and spleen, donor red blood cells coated with antibodies, typically IgG, undergo extravascular hemolysis (28). A delayed hemolytic transfusion reaction normally happens one to four weeks after transfusion (29,30).

***Transfusion-Associated Graft-versus-Host disease* (TaGVHD)**

A rare but fatal blood transfusion complication known as transfusion-associated graft-versus-host disease occurs, in which lymphocytes in the donated blood target the recipient's tissues, particularly those of immunocompromised recipients. This primarily affects the bone marrow, skin, and gastrointestinal system (31). The donor's lymphocytes attack host tissues due to his immunocompromised state (32).

TA-GVHD has also been observed in immunocompetent patients receiving blood from a family donor who is homozygous for a human leukocyte antigen (HLA) haplotype of which the recipient is heterozygous (32).

Post-transfusion purpura

A Rare, although life-threatening, transfusion reaction characterised by severe thrombocytopenia usually within two weeks of blood transfusion. It is associated with the development of alloantibodies to human platelet antigens (HPAs) (33).

Clinical Presentations and Management

Acute hemolytic transfusion reactions (AHTR) typically occur within the first hour after transfusion, though they can also occur later during the procedure or right afterwards. Patients suddenly report symptoms such as chest discomfort, shortness of breath, irritability, anxiety, fever with or without chills, flushing of the face and lower back, and intense pain. Anaphylactic shock can complicate the course, gastrointestinal symptoms as nausea and vomiting, and acute hemolysis may be followed by jaundice (34).

Laboratory findings include a Positive DAT (direct antiglobulin test) to determine whether a patient's red blood cells have been sensitised in the body with immunoglobulin, complement, or both. As intravascular hemolysis results in the presence of free hemoglobin in both plasma and urine, decreased haptoglobin levels and hyperbilirubinemia can be identified, so urinary hemoglobin, serum lactate dehydrogenase, bilirubin, and haptoglobin must be measured (35,36). In addition to this evaluation, an immune analysis that includes repeated crossmatching and blood grouping needs to be conducted (37).

Management starts with suspicion; the transfusion is discontinued immediately. Check airway, breathing, and circulation (ABC). Adequate Hydration is the initial therapy to achieve and maintain adequate blood pressure and renal blood flow. Pharmacologic treatment for hypotension must be done cautiously. Renal blood flow should be preserved by avoiding pressor medications such as epinephrine, norepinephrine, and high-dose dopamine; instead, dopamine at lower doses, 2 to 5 mcg/kg/minute IV, is typically utilised. It is essential to consult a nephrologist promptly, especially if no diuretic response occurs within 2 to 3 hours of starting treatment, as this may suggest acute tubular necrosis, in which early dialysis may be helpful (38,39). Anti-A and anti-B antibodies, as well as cytokines, can be eliminated via plasma exchange (40).

Ruxolitinib is a potent and rapid inhibitor of cytokine activity. It exerts its effects by obstructing the JAK-STAT signalling pathway. Targeting the JAK-STAT pathway leads to decreased production of several cytokines, particularly tumour necrosis factor alpha (TNF-α), through reduced activity of dendritic cells, T cells, and natural killer cells. (41,42) One published case report showed that treatment with ruxolitinib dramatically changed the rapidly progressive multiorgan failure in the patient, so it may be a life-saving treatment in patients with ABO-incompatible transfusion reaction, which follows a severe and catastrophic course (42).

Febrile non-hemolytic Transfusion reactions (FNHTR)

Febrile reactions in a clinical setting are characterised by a rise in temperature of more than 1 degree Celsius above the pretransfusion level, along with chills. They may also include headaches and lower back pain. Fever and chills can also present in severe hemolytic transfusion reactions, so it is essential to assess all febrile reactions for the possibility of an acute hemolytic transfusion reaction. The transfusion should be stopped immediately, and appropriate pharmacological treatment should be initiated (43).

Management is usually successful with antipyretics such as acetaminophen, antihistamines, and corticosteroids. Special leukoreduction filters are used for subsequent transfusions if the recipient has experienced more than one febrile reaction. Most hospitals use previously stored leuko-reduced (WBC-depleted) RBCs (44).

When TRALI occurs clinically, the patient will have a sudden onset of respiratory distress, fever, chest pain, hypotension, cyanosis, pulmonary oedema, and acute lung injury, which is characterised by a Ratio between arterial oxygen partial pressure (PaO2) and fractional inspired oxygen (FiO2) lower than 300 mmHg, accompanied by circulatory overload signs (27).

Management directed to discontinue the transfusion immediately with signs of respiratory distress, with active treatment of pulmonary oedema and hypoxia by ventilatory support (noninvasive or invasive) if indicated, along with other supportive therapy. A fatal outcome is less likely when prompt and comprehensive pulmonary assistance is provided (18,45). Diuretics are not contraindicated, and the hospital's blood bank or transfusion medicine service should be notified of such cases (20,46).

In a particular study, mechanical ventilation was needed in 78% of patients experiencing TRALI; pre-existing risk factors for acute lung injury have mainly impacted the clinical outcomes (47,48).

Allergic and anaphylactic Reactions (ATR)

They are common due to allergens present in the donor's blood. Reactions are typically mild, manifesting as hives, oedema, headaches, and sometimes dizziness. Allergic reactions usually present as maculopapular rash or urticaria without fever or hypotension. Anaphylactic reactions manifest with dyspnea, wheezing, bronchospasm, and hypotension (22.38). Anaphylactic reactions have been reported to be associated with anti-IgA in recipients who are IgA-deficient (49).

If an allergic reaction occurs, discontinue the transfusion immediately. Antihistamines usually control mild urticaria and itching. Hydrocortisone will be necessary for mild bronchospasm or a moderate allergic reaction with widespread urticaria. A severe anaphylactic reaction demands further intervention with epinephrine injection administered subcutaneously, normal saline IV, and the blood bank must be notified (20,49).

An antihistamine may be administered prophylactically to patients with a history of allergies or transfusion-related allergic reactions (50). Transfusions of washed red blood cells, washed platelets, or IgA-deficient plasma are necessary for patients with significant IgA deficiency (51).

Delayed immunologic Reactions

A delayed hemolytic transfusion reaction is usually mild and self-limiting. Patients may be asymptomatic or have a mild fever (30,52). On the other hand, more serious symptoms, such as jaundice, might develop. Lab results typically show a decreasing hematocrit along with a slight increase in lactate dehydrogenase and bilirubin levels. And a positive direct antiglobulin test (DAT) (53).

An unexplained drop in hemoglobin to the pretransfusion level occurring 1 to 2 weeks posttransfusion may be the only sign. Most delayed hemolytic reactions have a benign course and require no treatment; however, life-threatening hemolysis with severe anemia and renal failure may occur (29,53). Cross-matching compatible blood negative for the antigen; further transfusion is needed (30).

TA-GVHD usually manifests clinically within 4 to 30 days after transfusion, although the immunological process was probably initiated early (31).

Patients present with fever, which is typically linked with a widespread itchy maculopapular skin rash that often involves the face, trunk, and limbs, which can frequently advance to extensive erythroderma and toxic epidermal necrolysis, or an immune reaction targeting the gastrointestinal tract, resulting in anorexia, nausea, elevated transaminases, hepatomegaly, abdominal discomfort, and significant copious diarrhea. Diarrhea may be intense enough to lead to considerable electrolyte imbalances. TA-GVHD frequently affects the bone marrow, leading to thrombocytopenia or even pancytopenia (32,33).

The Diagnosis of TA-GVHD is usually challenging using skin, Liver, or bone marrow biopsy (34). The ideal management for TA-GVHD is prevention (54,55). Ruxolitinib was approved for the management of steroid-refractory acute graft-versus-host disease (GVHD) in patients aged 12 years or older (32,54). High-dose corticosteroids are used as the initial treatment in immunosuppressive therapy. Depending on the severity and progression of the disease, medications such as tacrolimus and cyclosporine may also be administered (53,54). The updated guidelines give optimism for the application of monoclonal antibodies in immunosuppression (55).

The most successful treatment for severe cases of TA-GVHD is emergency allogeneic hematopoietic stem cell transplantation (HSCT) (56).

As Prevention is the primary goal, the most effective method to prevent TA-GVHD is the use of irradiated blood products (57). The process of irradiating blood components with gamma or X-rays eliminates white blood cells, including functional T-cells, in the donated blood, thereby preventing them from attacking the recipient's tissues. This procedure is standard for certain blood transfusions, particularly those given to immunocompromised patients (58). Studies revealed no difference in efficacy between gamma and X-ray irradiation (59). The blood products that require treatment or irradiation include whole blood, packed RBCs, platelets, granulocytes, and non-frozen plasma. In contrast, fresh frozen plasma does not need to be irradiated (32,55).

More than 90% of TA-GVHD cases result in death because it causes the recipient's bone marrow to become aplastic, resulting in infections and bleeding (60,61).

In Patients with post-transfusion purpura, the transfusion must be discontinued immediately, and hydration with normal saline must be initiated. The patient's remaining blood products, both clotted and anticoagulated samples, should be delivered to the blood bank for analysis and reporting (62). Diagnosis is challenging because determining platelet antibodies frequently involves HPA typing of the patient's DNA to verify their specificity (63).

Treatment is usually with Intravenous immunoglobulin (IVIG), which is the drug of choice (63). With a response rate of 90% patients typically show a positive reaction to IVIG therapy within 48 hours. Corticosteroids are often given; plasma exchange was the first effective therapy and was considered for complex cases and for cases refractory to IVIG (64). Thrombocytopenia usually resolves within approximately 20 days if untreated (65)—platelet administration to patients with life-threatening bleeding (66).

There have been reports of a mortality rate between 10% and 20%, primarily due to intracranial bleeding. Post-transfusion purpura is a self-limited disease, and platelet counts recover within 20 days (67).

Non-Immunologic Transfusion Reactions

Transfusion Transmitted Infections

Transfusion of blood products carries an inherent risk of transmitting infections (TTI) due to their human-derived nature (68). In 1983, acquired immune deficiency syndrome caused by HIV was initially discovered (69). Testing for antibodies to both strains of HIV-1and HIV-2 is required, along with a Survey for blood donors regarding actions that could increase their risk of HIV infection (70).

Hepatitis B virus (HBV) was initially identified by Blumberg, where roughly 6% of patients who underwent multiple transfusions experienced transfusion-transmitted HBV (70). The discovery of HCV in 1989 led to the development of the HCV antibody test, and the incidence of transfusion-transmitted HCV sharply decreased as routine blood screening occurred (70).

In immunocompromised patients, Cytomegalovirus (CMV) can cause severe or life-threatening disease, so they should receive CMV-negative blood products based on antibody testing (71).

Human T-cell leukemia virus type I (HTLV-I) is associated with adult T-cell leukemia or tropical spastic paraparesis. Each unit of donated blood is tested for antibodies to HTLV-1 and HTLV-2 (71). Parvovirus B19 (B19V) is mainly spread through respiratory means but can also be transmitted through blood transfusions (72).

Blood intended for transfusions is usually collected in sterile conditions and regarded as germ-free. Yet, cases of transfusion-transmitted sepsis have been documented and confirmed by culture in approximately 1 in 100,000 recipients due to inadequate disinfection, as these bacteria typically originate from the normal skin flora. Except for cryophilic organisms such as Serratia, Pseudomonas, Acinetobacter, Yersinia, and E. coli, which can produce harmful endotoxin levels, refrigeration of red blood cells often inhibits bacterial growth (73).

Bacterial growth should be assessed in all RBC units. Because platelet concentrates are stored at room temperature, they have a greater potential for bacterial growth and endotoxin production if contaminated. Platelets are routinely tested for bacteria (68,73,74).

It is rare to be infected with syphilis through blood transfusion, as storing blood for ≥ 96 hours at 4 to 10° C kills the spirochete (75). Creutzfeldt-Jakob disease has never been reported to be transmitted by transfusion (76).

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Malaria parasites can be spread through blood transfusions, particularly via red blood cells obtained from donors who are asymptomatic and have parasitemia (77).

Clinical signs of transfusion-related sepsis include fever, rigors, hypotension, and shock. Investigations, including Gram stain and cultures, and septic markers, are recommended, with management of sepsis in accordance with local protocols (78).

Transfusion Associated Circulatory Overload (TACO)

Infusion of blood products in excess of the recipient's circulatory capacity leads to TACO, whether due to volume overload or rapid infusion rate (79).

TACO is characterised by pulmonary (cardiogenic) edema, which differentiates it from TRALI, which presents as noncardiogenic pulmonary edema (15,70). The transfusion of RBCs should be administered slowly with strict observation of the patient. The transfusion should be discontinued, and emergency heart failure treatment should commence if any sign of heart failure occurs (46,80)—typical treatment with Oxygenation, diuretics, and invasive or noninvasive mechanical ventilation. Patients at high risk of TACO (those with cardiac or severe renal insufficiency) are treated prophylactically with a diuretic therapy (80,81).

Massive transfusion

Massive transfusion refers to the transfusion of a blood volume that is equal to or greater than one full blood volume within a 24-hour period, which typically includes the administration of 10 or more units of whole blood or packed red blood cells (PRBCs) during that time frame. An ultra-massive transfusion is characterised by the use of more than 20 units of PRBCs over a 24- to 48-hour period (82).

In a massive Transfusion, the patient receives large volumes of resuscitation fluids, including packed RBCs (colloids) and crystalloids, leading to dilution of plasma clotting factors and platelets (83). This coagulopathy exacerbates the earlier coagulopathy caused by traumatic injury, leading to the fatal triad of acidosis, hypothermia, and bleeding (83,84). Complications of massive transfusion (table 2) are arrhythmias and hypothermia due to the rapid transfusion of large amounts of cold blood, which increases systemic vascular resistance, decreases cardiac output, and may lead to cardiac arrest (85,86).

The coagulation cascade is affected at temperatures below 33°C (87). Using blood warming devices can help prevent hypothermia, but complications can occur due to overheating, leading to potential RBC damage and hemolysis (88,89). Citrate metabolism is affected, and in the presence of hypothermia, leading to Citrate Toxicity with subsequent Hypocalcemia and hypomagnesemia, which can significantly occur in patients with liver failure (90). Hyperkalemia can be a complication, especially in patients with renal disease, due to the increased potassium content of stored RBCs. At the same time, Hypokalemia may occur approximately 24 hours after transfusion of older RBCs or due to citrate toxicity, resulting in metabolic alkalosis (90,91).

Massive transfusion protocols recommend administering fresh frozen plasma and platelets earlier in the resuscitation process, before the onset of coagulopathy, to reduce mortality (91).

Table 2: Massive Transfusion Complications

Massive Transfusion Complications	
Hypothermia	Metabolic acidosis
Dilutional coagulopathy	Hyperkalemia and hypokalemia
Hypocalcemia	Citrate toxicity
Hypomagnesaemia	TACO and TRALI

Iron overload related to transfusions is directly proportional to the frequency of transfusions. Patients with iron overload typically have elevated serum iron levels and a low total iron-binding capacity (TIBC) (92). Deferoxamine is the preferred treatment for iron overload and can be given as a continuous intravenous or subcutaneous infusion (92).

CONCLUSION

Blood transfusions are now an essential intervention in modern medical practice, and all healthcare workers should be familiar with transfusion reactions. Some reactions can be severe and lead to death, while many transfusion reactions are mild. Anaphylactic reactions from a blood transfusion are infrequent but often result in death. Understanding how to identify transfusion reactions and to manage the patient in a timely and appropriate manner ensures the patient receives optimal treatment. Any symptom that arises within 24 hours post blood transfusion must be regarded as a transfusion reaction until proven otherwise.

DECLARATIONS

Data Availability Statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department Concerned

Consent for publication

Approved

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Not applicable

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTION

MOHAMED IBRAHIM SHOAIB and MAI MOSSAD ELMEISSERY *contributed equally in drafting the manuscript, Manuscript Review, Concept of the work, and design.*

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