COMPLICATIONS OF BLOOD TRANSFUSION

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Complications of Blood Transfusion

Immune Complications
- Hemolytic Reactions
  - Acute
  - Delayed
- Non-Hemolytic Immune Reactions

Infectious Complications
- Viral Infections
- Bacterial Infections
- Parasitic Infections
• The most common complications of transfusion are febrile nonhemolytic.
• The most serious complications are acute hemolytic reaction due to ABO incompatible transfusion and transfusion-related acute lung injury, which have very high mortality rates.
1. Hemolytic Reactions

Hemolytic reactions usually involve specific destruction of the transfused red cells by the recipient’s antibodies. Less commonly, hemolysis of a recipient’s red cells occurs as a result of transfusion of red cell antibodies. Incompatible units of platelet concentrates, FFP, clotting factor concentrates, or cryoprecipitate may contain small amounts of plasma with anti-A or anti-B (or both) alloantibodies. Transfusions of large volumes of such units can lead to intravascular hemolysis.

Hemolytic reactions are commonly classified as either acute (intravascular) or delayed (extravascular).
Acute Hemolytic Reactions

The most common cause is **misidentification of a patient, blood specimen, or transfusion unit**, a risk that is not abolished with autologous blood transfusion.

In anesthetized patients, an acute hemolytic reaction may be manifested by 1. a **rise in temperature** 2. **unexplained tachycardia** 3. **hypotension** 4. **hemoglobinuria** 5. **diffuse oozing in the surgical field**, or a combination of these findings.

DIC, shock, and kidney acute failure can develop rapidly. The severity of a reaction often depends upon **the volume of incompatible blood** that has been administered.
Management of hemolytic reactions can be summarized as follows:

1. If a hemolytic reaction is suspected, the transfusion should be stopped immediately and the blood bank should be notified.

2. The unit should be rechecked against the blood slip and the patient’s identity bracelet.

3. Blood should be drawn to identify hemoglobin in plasma, to repeat compatibility testing, and to obtain coagulation studies and a platelet count.

4. A urinary bladder catheter should be inserted, and the urine should be checked for hemoglobin.

5. Forced diuresis should be initiated with mannitol and intravenous fluids, and with a loop diuretic if necessary.
Delayed Hemolytic Reactions

also called extravascular hemolysis—is generally mild and is caused by antibodies to non-D antigens of the Rh system or to foreign alleles in other systems such as the Kell, Duffy, or Kidd antigens.

By the time significant amounts of these antibodies have formed (weeks to months), the transfused red cells have been cleared from the circulation. Moreover, the titer of these antibodies subsequently decreases and may become undetectable.

Reexposure to the same foreign antigen during a subsequent red cell transfusion, however, triggers an anamnestic antibody response against the foreign antigen. The hemolytic reaction is therefore typically delayed 2 to 21 days after transfusion, and symptoms are generally mild, consisting of malaise, jaundice, and fever. The patient’s hematocrit typically fails to rise, or rises only transiently, in spite of the transfusion and the absence of bleeding.

The serum unconjugated bilirubin increases as a result of hemoglobin breakdown.
2. Nonhemolytic Immune Reactions

1. Febrile Reactions
2. Urticarial Reactions
3. Anaphylactic Reactions
4. Transfusion-Related Acute Lung Injury
5. Transfusion-Associated Circulatory Overload
6. Graft-Versus-Host Disease
7. Post-Transfusion Purpura
8. Transfusion-Related Immunomodulation
Febrile Reactions

White cell or platelet sensitization is typically manifested as a febrile reaction. Such reactions are relatively common (1–3% of transfusion episodes) and are characterized by an increase in temperature without evidence of hemolysis. Patients with a history of repeated febrile reactions should receive leukoreduced transfusions only.
Urticarial Reactions

Urticarial reactions are usually characterized by erythema, hives, and itching without fever. They are relatively common (1% of transfusions) and are thought to be due to sensitization of the patient to transfused plasma proteins. Urticarial reactions can be treated with antihistaminic drugs (H1 and perhaps H2 blockers) and steroids.
Anaphylactic Reactions

Anaphylactic reactions are rare. These severe reactions may occur after only a few milliliters of blood has been given, typically in IgA-deficient patients with anti-IgA antibodies who receive IgA containing blood transfusions. Such reactions require treatment with epinephrine, fluids, corticosteroids, and H1 and H2 blockers. Patients with IgA deficiency should receive thoroughly washed packed red cells, deglycerolized frozen red cells, or IgA-free blood units.
Transfusion-Related Acute Lung Injury

It presents as acute hypoxia and noncardiac pulmonary edema occurring within 6 h of blood product transfusion.

It occur with transfusion of any blood component, but especially platelets and FFP. Treatment is similar to that for acute respiratory distress syndrome, with the important difference that TRALI may resolve within a few days with supportive therapy.

The incidence of TRALI, until recently the leading cause of transfusion-related death, has markedly declined with the recognition that the presence of HLA antibodies in donor plasma is the principal TRALI risk factor.
Transfusion-Associated Circulatory Overload

Transfusion-associated circulatory overload (TACO) occurs when blood products are administered at a rate greater than the patient’s cardiac output, usually in a massive haemorrhage resuscitation scenario. This is most likely to occur when the provider administering blood products has not recognized that the source of bleeding has been successfully controlled.

TACO has replaced TRALI as the leading transfusion-related risk for trauma patients.
Post-Transfusion Purpura

It is a potentially fatal thrombocytopenic disorder that occurs, rarely, following blood or platelet transfusion. It results from development of platelet alloantibodies that destroy the patient’s own platelets. The platelet count typically drops precipitously 5 to 10 days following transfusion. Treatment may include intravenous IgG and plasmapheresis.
INFECTIOUS COMPLICATIONS
1. Viral Infections

A. Hepatitis (B&C)
Most acute cases are anicteric. **Hepatitis C** is the more serious infection; most cases progress to chronic hepatitis, with **cirrhosis** developing in 20% of chronic carriers and **hepatocellular carcinoma** developing in up to 5% of chronic carriers.

B. Acquired Immunodeficiency Syndrome (AIDS)
All blood is tested for the presence of anti-HIV-1 and anti-HIV-2 antibodies. The requirement for donor blood testing by the U.S. Food and Drug Administration (FDA) has decreased the risk of transfusion-transmitted HIV.
MASSIVE BLOOD TRANSFUSION

It is most often defined as the need to transfuse the patient’s total estimated blood volume in less than 24 h, or one-half the patient’s total estimated blood volume in 1 h. For most adult patients, the total estimated blood volume is the equivalent of 10 to 20 units. The approach to massive transfusion (and to lesser degrees of transfusion) elucidates that the outcomes have improved with concurrent transfusion of packed red cells, FFP, and platelets to avoid dilutional coagulopathy.
1. Coagulopathy

The most common cause of nonsurgical bleeding following massive blood transfusion is dilutional thrombocytopenia, which is caused by platelet loss out of the body and platelet dilution with replaced red cells and crystalloids. This thrombocytopenia is transient and the platelet count will usually return to normal once the patient's circulation equilibrates. Although clinically significant dilution of coagulation factors may also occur.
2. Citrate Toxicity

Calcium binding by the citrate preservative can rise in importance following transfusion of large volumes of blood or blood products. Clinically important hypocalcemia, causing cardiac depression, will not occur in most normal patients unless the transfusion rate exceeds 1 unit every 5 min, and intravenous calcium salts should rarely be required in the absence of measured hypocalcemia.

Because citrate metabolism is primarily hepatic, patients with hepatic disease or dysfunction (and possibly hypothermic patients) may demonstrate hypocalcemia and require calcium infusion during massive transfusion, as may small children and others with relatively impaired parathyroid–vitamin D function.
3. Hypothermia

Massive blood transfusion is an absolute indication for warming all blood products and intravenous fluids to normal body temperature.

Ventricular arrhythmias progressing to fibrillation often occur at temperatures close to 30°C, and hypothermia will hamper cardiac resuscitation. The customary use of rapid infusion devices with efficient heat transfer capability has decreased the incidence of transfusion-related hypothermia.
4. Acid–Base Balance

Although stored blood is acidic due to the citric acid anticoagulant and accumulation of red cell metabolites (carbon dioxide and lactic acid), metabolic acidosis due to transfusion is uncommon because citric acid and lactic acid are rapidly metabolized to bicarbonate by the normal liver. However, in the situation of massive blood transfusion, acid–base status is largely dependent upon tissue perfusion, rate of blood transfusion, and citrate metabolism.
5. Serum Potassium Concentration

The extracellular concentration of potassium in stored blood steadily increases with time. The amount of extracellular potassium transfused with each unit is typically less than 4 mEq per unit. Hyperkalemia can develop regardless of the age of the blood when transfusion rates exceed 100 mL/min.
Thank you