Blood Transfusion Reactions

Maggie Craig, MD
UT Southwestern Medical Center
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Types of Blood Transfusion Reactions

1. Febrile, non-hemolytic transfusion reaction
2. Bacterial infections
3. Viral infections
4. Acute hemolytic reaction
5. Transfusion-associated graft vs. host disease
6. Anaphylactic reaction
7. Transfusion-related acute lung injury (TRALI)
8. Volume overload (TACO)
9. Iron overload
10. Delayed hemolytic reaction
Febrile Non-Hemolytic Transfusion Reaction

- Most common blood transfusion reaction
- Etiology – Cytokines in donor WBCs cause the symptoms
- Incidence – Approx 1% of blood tx. Increases with prior blood tx or with multiple pregnancies
- Symptoms – fever, chills, rigors, dyspnea
- Fever – defined as an increase in 1.0 ° Celsius or 2 ° F
- Clinically benign – must rule-out hemolytic and septic transfusion reactions
- Symptoms begin during blood tx or up to 2 hrs after blood tx
Febrile Non-Hemolytic Transfusion Reaction
Febrile Non-Hemolytic Transfusion Reaction

- Prevention: universal leukoreduction of RBCs and platelets
- Febrile, non-hemolytic transfusion reaction also occurs with platelets
Bacterial Infections

- Risk is highest with platelets since they are stored at room temperature (Mortality is 1:50,000) Bacterial infection from platelets as high as 1:700.
- Risk for RBC sepsis is 1:500,000, difficult to treat, may be fatal.
- Common pathogens for RBC contamination are:
  - Yersinia, Serratia, Pseudomonas (all can grow in cold, stored blood)
  - Visually inspect the blood to look for clots or color change
  - Symptoms: fever, rigors, hypotension, chills, profound shock
  - Diagnosis: gram stain the remaining amount of blood in the tubing/bag
Viral Infections

• Common viral pathogens include: Hep B, Hep C, HIV, variant CJD (mad cow).
• Hepatitis B risk per unit of transfused blood – 1 in 200,000
• Hep C risk – 1 in 2 million
• HIV risk – 1 in 2 million
• Creutzfeldt-Jakob Disease – very low risk. Persons exposed to vCJD are not allowed to give blood
Acute Hemolytic Reaction

- Usually related to ABO incompatibility
- Usually occurs within minutes of starting a transfusion
- Most common cause is clerical error (see picture)
- Host antibodies rapidly destroy the donor RBCs. Most severe reactions occur when a patient with O-blood type receives group A RBCs
Pathophysiology of AHR

- Recipient has IgM anti-A,B antibodies circulating, receives the wrong blood unit containing Type AB blood
- Anti-A,B antibodies in the recipient activate the complement cascade C3a and C5a → inflammatory cytokines are released (IL-1, IL-6, TNFα) → fever, chills, dyspnea
- Antigen-antibody complexes can also cause release of bradykinin → increased capillary permeability and arteriolar vasodilation (pulm edema, hypotension).
- Factor 12 is activated → thrombin is formed, causing clots, DIC. Renal failure results from hypotension, thrombin clots, antigen-antibody complex deposition, ischemia.
Main symptoms of Acute hemolytic reaction

- **Systemic**
  - Chills
  - Fever

- **Vascular**
  - Hypotension
  - Uncontrollable bleeding

- **Heart**
  - Increased heart rate

- **Chest**
  - Constricting pain

- **Transfused vein**
  - Heat sensation

- **Urinary**
  - Hemoglobulinuria
  - Hyperbilirubinemia

- **Lumbar region**
  - Pain
Treatment of AHR

- Stop the transfusion
- Support the blood pressure with fluid/pressors
- Consider giving mannitol and lasix to continue diuresis
- Give plenty of IV hydration (normal saline)
- Obtain blood sample and look at the centrifuged blood for pink hemoglobin
- Obtain urine dipstick and assess for blood
- Send blood for Direct Coomb’s Test
- Bicarbonate has not been proven effective
Acute Hemolytic Reaction

- Acute hemolytic reactions may be either immune-related or non-immune related
- **Immune-mediated** reactions are caused by IgM anti-A, anti-B, or anti-A,B (ABO incompatibility)
- **Non-immune mediated** acute hemolytic reactions occur when RBCs have been damaged prior to transfusion (physically damaged)
- **Non-immune mediated** AHR causes hemoglobinuria and hemoglobinemia without other symptoms
Hematuria vs. Hemoglobinuria
Transfusion-Related GVHD

- Most GVHD (graft vs. host disease) occurs after stem cell transplantation
- It is rarely associated with blood transfusions
- Occurs only in those patients who are immunosuppressed from congenital deficiency or hematologic malignancy
- Uniformly fatal when it occurs
- Prevention: irradiated blood products
Anaphylactic Reaction to Blood Transfusion

- Most common in IgA deficiency patients (anti-IgA antibodies present in the recipient)
- Incidence: 1:20,000
- Other allergic reactions to soluble blood antigens are possible, and are benign
- Symptoms: rash, urticaria, pruritus. These are IgE mediated. For rash without fever, you do NOT have to stop the blood transfusion, just give Benadryl to help pruritus.
- Prevention: washed RBCs, plasma depleted transfusions
Transfusion-associated acute lung injury (TRALI)

- Most common cause of transfusion-related mortality today
- Acute respiratory distress, fever, non-cardiogenic pulmonary edema, hypotension
- Most patients recover within 96 hours, mortality is less than 10%
- Incidence: 1:2000 transfusions
TRALI
TRALI

- Mechanism of the capillary leak in TRALI is not known
- WBC antigens and cytokines have been implicated
- Symptoms: hypoxemia, fever, hypotension (usually), bilateral pulmonary edema that develop within 6 hrs of blood tx
- JVD, rales, dilated PA on CXR rule-out TRALI
Transfusion-associated Circulatory Overload (TACO)

- Acute pulmonary edema and respiratory distress due to volume overload
- Patients with underlying poor cardiac function are at greater risk
- Presents as acute respiratory distress, like TRALI, but will not have transient leukopenia like TRALI, and will have evidence on CXR of heart overload (e.g., enlarged heart, dilated PA, elevated JVD, rales)
- Patient more likely to have HYPERtension with TACO, HYPOtension with TRALI
- Treatment: oxygen, diuretics (good for TACO, bad for TRALI), phlebotomy, placing patient in sitting position
Iron Overload

• This usually occurs in patients who receive chronic blood transfusions (beta-thalassemia)
• Acutely, it only occurs in patients getting more than 12-20 units of blood at one time
• In patients with beta-thalassemia, increased iron absorption occurs and overloads the body’s ability to store the iron. The iron is deposited in the endocrine glands (pancreas), myocardium, parathyroids, pituitary, liver.
• In the myocardium, it leads to early death
• Treatment: iron chelation therapy
Delayed Hemolytic Reaction

- Occurs more than 24 hours after a blood transfusion
- Etiology: a secondary immune response from the recipient’s antibodies to the donor’s RBC antigens
- Risk: increases in patients receiving multiple transfusions
- Severity can range from mild to life-threatening
- Symptoms: fever, lower than expected Hgb level, jaundice, urobilinogen in the urine
Delayed Hemolytic Reaction

- Evidence of hemolysis within a week of transfusion is characteristic
- Fever is usually present
- Hemoglobinuria and hemoglobinemia are rare
- On a peripheral smear, spherocytes, reticulocytosis are seen. Higher unconjugated bilirubin and LDH are seen on blood chemistry analysis.
Delayed Hemolytic Reaction
What is a Coomb’s Test?

- **Direct Coomb’s test** is when the recipient RBCs possess antibodies on their surface. You mix antiserum with the recipient RBCs and agglutination occurs. Positive in autoimmune hemolytic anemia, acute hemolytic transfusion reaction.

- **Indirect Coomb’s test** is when the recipient’s plasma contains antibodies. You mix the recipient’s serum with normal RBCs and the antiserum. Agglutination occurs. This is positive in alloimmunization, or tx of incompatible blood type.
The End

• It is a rough road that leads to the heights of greatness. (Seneca)

• Success is how high you bounce when you hit bottom. (Gen. George Patton)