TRANSFUSION MEDICINE—LECTURE 3

Complications

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OUTLINE:

- Non-Infectious Complications
  - Allergic reactions
  - Hemolytic reactions
  - Respiratory reactions
  - Miscellaneous reactions

- Infectious Complications
  - Viral complications
  - Bacterial complications
  - Protozoal complications
  - "Other" complications involving infectious agents

OBJECTIVES:

- Be familiar with the most common symptoms of the various transfusion reactions (allergic, anaphylactic, febrile, acute hemolytic, delayed hemolytic, TRALI, TACO) and the appropriate treatment
- Describe the treatment for acute hemolytic transfusion reactions
- Describe the mechanisms suggested for TRALI
- Describe the pathophysiology of transfusion-associated graft-versus-host disease and the patients at risk for this complication and its prevention
- Describe neonatal immune thrombocytopenia (NAIT) and post transfusion purpura (PTP)
- Know the various infectious agents known to be transmitted by transfusion and their approximate risk
- Be familiar with the risk of bacterial contamination of blood products and the possible patient symptoms associated with this complication

REFERENCES:

Extensive notes are provided to accompany the lectures in Transfusion Medicine

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Transfusion Medicine
LECTURE 3
Complications

Jerry E. Squires MD, PhD

Transfusion Complications--Classification

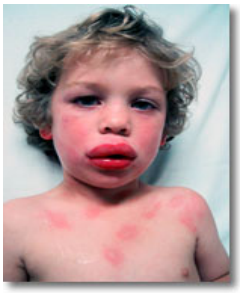
- **Non-Infectious Complications**
  - *Classified by predominant symptom*
    - **Allergic**
      - Simple allergic txf. rx.
      - Anaphylactic txf. rx.
    - **Hemolytic**
      - Acute hemolytic txf. rx.
      - Delayed hemolytic txf. rx.
    - **Respiratory**
      - Transfusion-related acute lung injury (TRALI)
      - Transfusion-associated circulatory overload (TACO)
    - **Miscellaneous**
      - Febrile nonhemolytic txf. rx.
      - Graft versus host disease
      - Platelet-associated reactions
      - Iron Overload
- **Infectious Complications**
  - *Classified by infectious agent*
    - **Virus**
      - Hepatitis B
      - Hepatitis C
      - HIV
      - HTLV
    - **Bacteria**
      - Bacterial contamination
      - Syphilis (*T. pallidum*)
    - **Protozoa**
      - Malaria (*Plasmodium sp.*)
      - Chagas (*T. cruzi*)
    - **Other**
      - Prions (variant Creutzfeld-Jacob Disease)

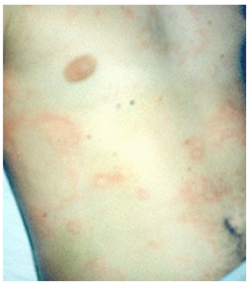
Allergic and Anaphylactic Reactions

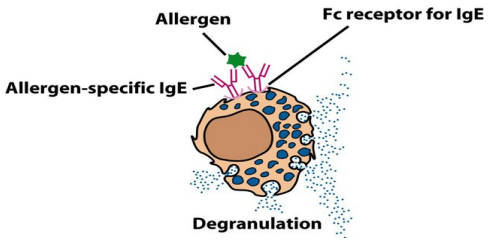
- Allergic reactions are among the most common adverse reactions to transfusion
  - Simple allergic reactions complicate 1-3% of transfusions
- In evaluating allergic-type reactions, it is reasonable to consider the simple allergic reaction and the more serious anaphylactic reaction (*severe hypotension*) as a spectrum of allergic symptoms caused by fundamentally similar mechanisms
- Starting with simple allergic reactions…

Allergic Transfusion Reactions

- **Recognition:**
  - Urticaria (Hives)
  - Pruritis
  - Flushing
  - Localized Angioedema
  - Wheezing (laryngeal edema)
- **Pathophysiology:**
  - Most allergic transfusion reactions are examples of *Type I hypersensitivity reactions*
  - An allergen (usually some transfused plasma protein) combines with a pre-formed IgE antibody on the surface of mast cells and basophils of the recipient
  - Mast cells and basophils are then activated to release various anaphylatoxins







Allergic Transfusion Reactions

- **Treatment:**
  - Suspend transfusion
  - Treat with antihistamine; prednisone can be added if antihistamine alone is insufficient
  - Restart transfusion if hives resolve
- **Prevention:**
  - Repeated allergic reactions recur in only a minority of patients
  - 60-80% of patients receive an antihistamine (25-50 mg diphenhydramine) prior to transfusion in the U.S. However, there is no evidence that this significantly reduces the risk of allergic reactions.
  - Treatment with an antihistamine when an allergic reaction occurs is generally helpful in resolving the symptoms.
  - For severe recurrent allergic reactions or reactions unresponsive to antihistamine treatment, red cell and platelet products can be washed to remove residual plasma

Anaphylactic Reactions

The more severe version of allergic reactions!!

- **Recognition:**
  - These reactions can have any/all of the symptoms associated with simple allergic reactions

PLUS

  - Severe Hypotension (Shock)
- **Pathophysiology:**
  - Most anaphylactic reactions resulting from transfusion are IgE-mediated class I hypersensitivity reactions—as described previously
  - However, several specific situations have been associated with the development of anaphylactic reactions:
    - IgA antibodies in patients with severe IgA deficiency (serum IgA <0.05 mg/dL)
    - Pre-existing antibodies to certain other serum proteins (haptoglobin, albumen, transferrin, etc.)
    - Transfusion of allergens in donor blood products (e.g. penicillin) to a patient allergic to that allergen

Anaphylactic / Anaphylactoid Reactions

- **Treatment:**
  - Stop transfusion
    - In patients with anaphylactic symptoms, the transfusion may **NOT** be re-started
  - Maintain oxygenation in patients with respiratory distress (O_2 by nasal cannula). Aminophylline for bronchospasm)
  - Stabilize hypotension
    - Crystalloids (normal saline)
    - Epinephrine
    - Dopamine if hypotension is unresponsive
- **Prevention:**
  - If due to IgA deficiency (or other plasma protein), red cell and platelet products can be washed to deplete plasma proteins
  - Products, particularly plasma, can be collected from IgA deficient donors
  - NOTE: a patient who is IgA deficient but has **NO** anti-IgA antibodies and no history of anaphylactic reactions does not require the transfusion of washed or IgA deficient products

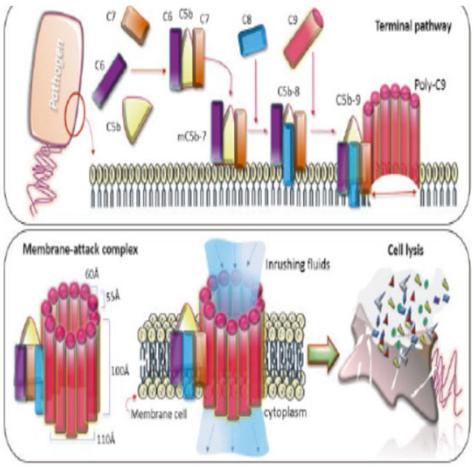
Hemolytic Transfusion Reactions (HTR)

- Typically, hemolytic transfusion reactions occur when antigen-positive RBCs are transfused to a patient who has an alloantibody (*i.e.* an antibody to an antigen that the patient does not have).
- Hemolytic transfusion reactions are typically classified as acute or delayed
  - **Acute**—Hemolysis occurs within 24 hours of transfusion and the antibody is generally present at the time of transfusion
  - **Delayed**—hemolysis occurs more than 24 hours after transfusion; the alloantibody is generally NOT detectable at the time of transfusion but develops later
- A second way of thinking about hemolytic transfusion reactions is by the "site" of hemolysis which depends on the number and density of RBC antigens sites, the immunoglobulin class and subclass, and the activation of complement:
  - **Intravascular hemolysis**
  - **Extravascular hemolysis**

Hemolytic Transfusion Reactions:

Intravascular Hemolysis

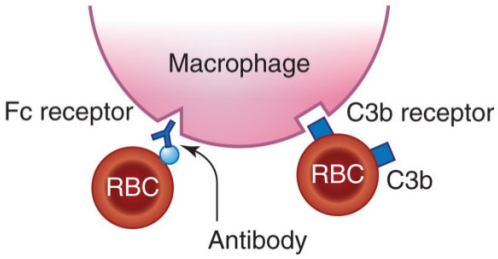
- Intravascular Hemolysis
  - Results in the intravascular lysis of RBCs
  - Intravascular lysis requires the activation of complement, generally through the classical pathway.
  - IgM antibodies are potent activators of complement since a single molecule of IgM can bind C1q and activating the complement cascade.
  - IgG is usually less likely to activate complement; relatively few classes of IgG can activate complement by binding to C1q if the antigen sites are sufficiently close together
  - The activation of C1q,initiates the complement cascade leading to the formation of the **membrane attack complex (MAC)** and the lysis of RBCs



Hemolytic Transfusion Reactions:

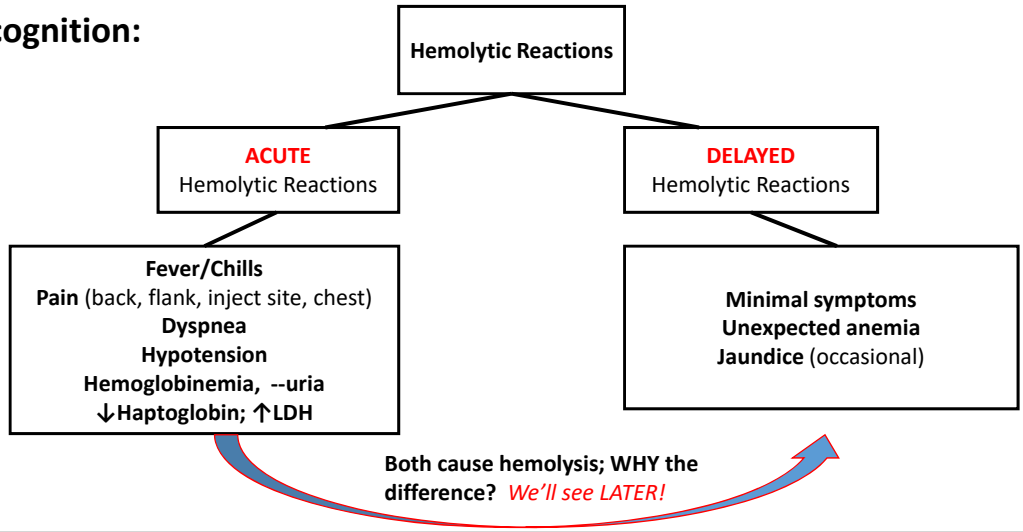
Extravascular Hemolysis

- Extravascular Hemolysis
  - When the complement cascade cannot be activated the binding the IgG to the surface of RBCs, opsonizes the RBCs which results in the removal of RBCs by the reticuloendothelial cells of the spleen and liver
  - Phagocytic cells of the reticuloendothelial system (RES) bind to the Fc of the IgG antibodies or to C3b attached to the RBCs



Hemolytic Transfusion Reactions

- **Recognition:**

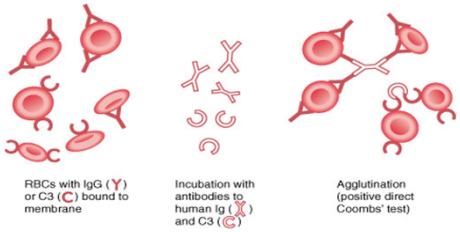


Acute Hemolytic Reactions

- Laboratory Evaluation:
  - Clerical check
    - The majority of acute hemolytic transfusion reactions are caused by clerical errors (wrong blood to wrong patient; mis-identified blood samples for cross match)
  - Recipient blood sample
    - Hemolysis (visual)
    - Confirm patient ABO
    - Direct Antiglobulin Test
  - Blood product
    - Re-type blood product

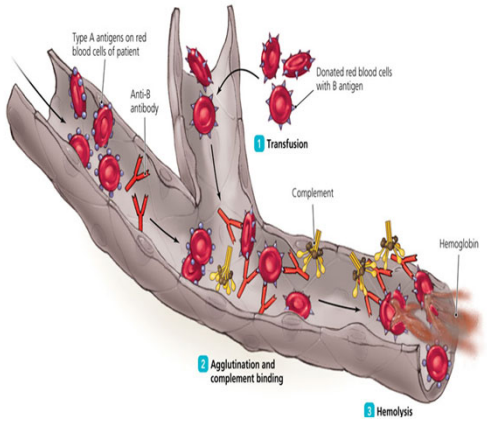
- Hemolysis:

- Direct Antiglobulin Test (DAT; Coombs)



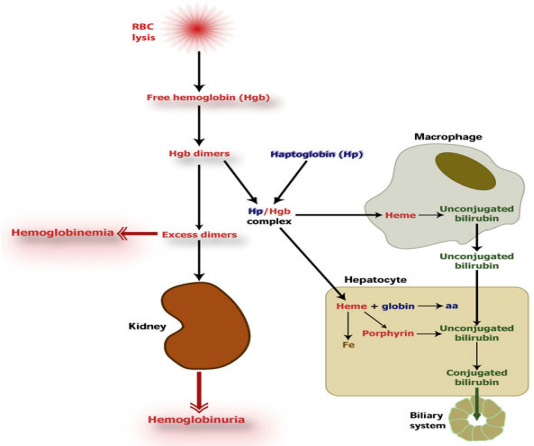
Acute Hemolytic Reactions

- **Pathophysiology:**
  - Most cases of Acute Hemolytic Transfusion Reactions are due to the transfusion of ABO-incompatible red cells to patients with ***pre-existing*** anti-A or anti-B
  - Anti-A and anti-B are usually IgM which can efficiently fix **complement** which can lead to the development of the membrane attack complex causing ***intravascular hemolysis*** resulting in:
    - Hemoglobinemia
    - Hemoglobinuria
    - ↑ Bilirubin
  - Some IgG antibodies can also fix complement as well and cause *intravascular hemolysis*; more often IgG antibodies cause ***extravascular hemolysis*** with RBC coated with IgG removed from circulation by the reticuloenothelial system (principally the spleen). Mainly causing:
    - ↑ Bilirubin



Acute Hemolytic Reactions

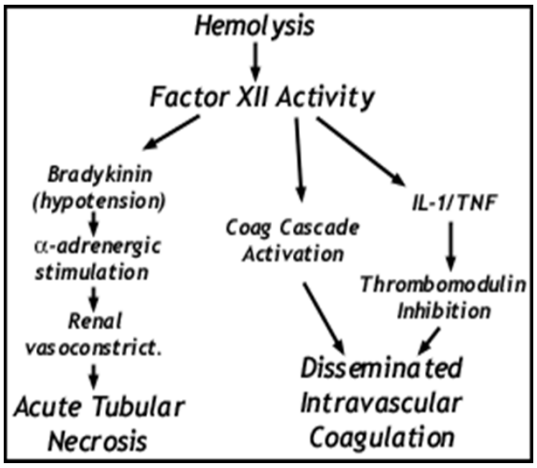
- Pathophysiology:
  - Looking at the pathophysiology more comprehensively to explain the signs and symptoms:



Acute Hemolytic Reactions

Complications

- The most significant threats of AHTR are the complications:
  - **Renal Failure (Acute Tubular Necrosis)**
  - **Disseminated Intravascular Coagulation (DIC)**
- There are several contributory mechanisms that probably play a role in the development of these complications:
  - Antigen-antibody complexes that precipitate hemolysis may also activate Factor XII
  - Factor XII may cause hypotension through the production of bradykinin
  - Activated Factor XII can also activate the coagulation cascade, TNFα, and IL-1
  - This can ultimately lead to the development of DIC
- It is important to realize that this represents only the "tip-of-the-iceberg" in understanding the complications of HTRs since a variety of cytokines (IL-1, IL-6, IL-8, etc. also may play a role in the development of these complications

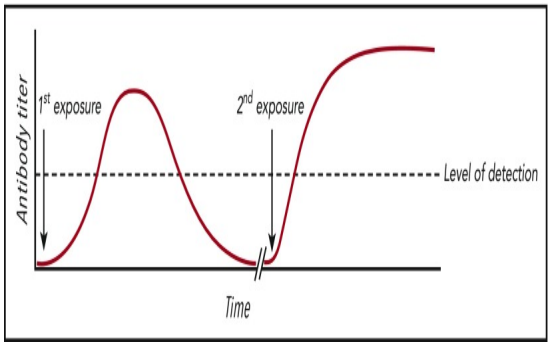


Acute Hemolytic Transfusion Reactions

- Treatment:
  - Prompt recognition of the symptoms of AHTR is critical (overall the consequences of AHTR are more severe as more incompatible blood is transfused!!)
  - If a suspected AHTR is recognized:
    - STOP transfusion immediately
    - Maintain venous access; infuse normal saline
    - Maintain urine output
      - Diuretics may be useful (furosemide)
    - Maintain blood pressure
    - Monitor coagulation status
- Prevention:
  - Most AHTR result from the transfusion of ABO-incompatible blood.
  - This is largely due to errors in patient identification (wrong patient receives blood labeled for someone else) or the mis-identification of the blood sample intended for cross-matching the patient (~1:4000 blood samples are labeled with the wrong patient name!!!)

Delayed Hemolytic Transfusion Reactions

- AHTR are caused by the presence of a ***pre-existing*** red cell antibody (e.g. anti-A; anti-B)
- In **delayed hemolytic transfusion reactions** (DHTR), the red cell antibody is not present (or is undetectable) at the time of transfusion
- In DHTR the red cell antibody develops in the recipient during the 7-21 days ***after*** the transfusion (often an anamnestic response) *and is caused by exposure to foreign red cell antigens on the transfused red cells.*
  - Alloantibodies to blood group antigens tend to rise after the first exposure to a foreign antigen, but then decrease over time becoming undetectable until the patient is re-exposed to the foreign antigen causing an anamnestic response
- The development of this antibody results in the destruction of the remaining transfused red cells (*which bear the antigen*)



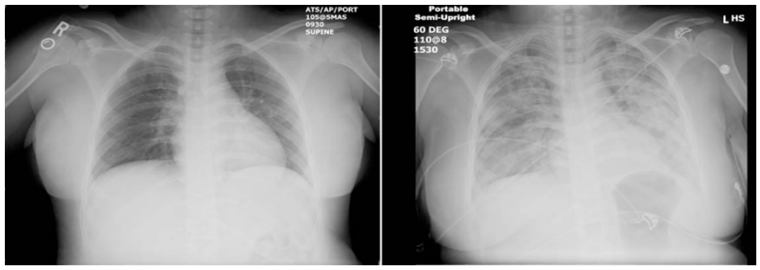
Delayed Hemolytic Transfusion Reactions

- Treatment:
  - Often treatment is unnecessary with the only symptoms being unexpected anemia and occasionally jaundice
  - Transfusion might be required
- Prevention:
  - The patient's transfusion history is always reviewed by blood bank staff to determine if there have been previous transfusions, previous red cell antibodies detected, or previous transfusion reactions.
  - DHTR are often unavoidable as the antibody is undetectable at the time of pre-transfusion testing and previous transfusions occur at another hospital

Transfusion Related Acute Lung Injury (TRALI)
Transfusion Associated Circulatory Overload (TACO)

- The next two transfusion reactions may seem initially to be very similar with respect to their presenting symptoms (RESPIRATORY DISTRESS accompanied by a Chest X-Ray showing BILATERAL PULMONARY INFILTRATES)

Pre-Txf.



Post-Txf.

- But these two distinct reactions are quite different in etiology and treatment

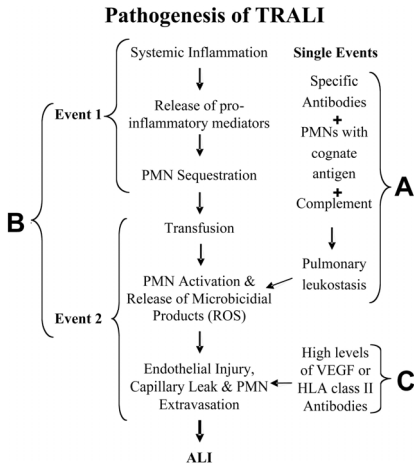
TRALI / TACO—Comparing Symptoms!

- **Transfusion Related Acute Lung Injury:**
- ***Presenting Symptoms***
  - Dyspnea
  - Bilateral Pulmonary Infiltrates on CXR
- ***Distinguishing Symptoms***
  - Fever/Chills
  - Hypotension
  - CVP: normal to low

- **Transfusion Associated Circulatory Overload:**
- ***Presenting Symptoms***
  - Dyspnea
  - Bilateral Pulmonary Infiltrates on CXR
- ***Distinguishing Symptoms***
  - Orthopnea
  - Hypertension
  - CVP elevated
  - ↑ BNP (Brain Naturetic Peptide)
    - *This is not a reliable indicator of TACO, but it is helpful when elevated!*

Transfusion Related Acute Lung Injury (TRALI)

- The pathogenesis of TRALI is complex and is thought to be precipitated in 3 ways:
  - Single clinical event (**A**):
    - Infusion of **donor antibodies directed against recipient leukocytes**
    - This mechanism is most certain, but it only accounts for ~85% of the cases of TRALI
  - "Two-hit" mechanism (**B**):
    - First "hit" refers to the clinical condition of the patient causing pulmonary endothelial activation and adherence of PMNs
    - Second "hit" is the transfusion of biologic response modifiers
  - Agents that cause endothelial "fenestration" in neutropenic patients (**C**):
    - High levels of VEGF or
    - High levels of HLA Class II antibodies



Transfusion Related Acute Lung Injury (TRALI)

- **Treatment:**
  - Stop transfusion
  - Prompt respiratory support
  - Intubation and mechanical ventilation, if necessary
  - Vasopressors for hypotension
  - Clinical improvement generally occurs within 3 – 5 days; resolution of symptoms is generally complete
  - Diuretics NOT indicated
- **Prevention:**
  - Minimize the use of donors who have increased chance of having anti-leukocyte antibodies
    - Multiparous females have increased probability of having anti-leukocyte (HLA) antibodies
    - Produce transfusable plasma and platelets from predominantly male donors (*these are the 2 products that contain the MOST plasma and hence the greatest chance of having large amounts of HLA-antibodies)*

Transfusion-Associated Circulatory Overload (TACO)

- Pathophysiology:
  - Manifestation of simple volume overload
  - Increased CVP
  - Congestive heart failure
- Treatment:
  - Stop transfusion
  - Manage symptoms (supplemental oxygen, etc.)
  - Diuretics indicated
- Prevention:
  - Carefully manage volume in susceptible patients, for example:
    - Elderly
    - Cardiac disease
    - Congestive heart failure
    - Infants / neonates
  - Slow infusion rate to 1.0 mL/kg/hour (usual rate 2.0 – 2.5 mL/kg/hour)

Miscellaneous Transfusion Reactions

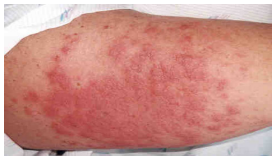
- In the next series of slides, we will be discussing several unrelated transfusion reactions including:
  - Febrile, Nonhemolytic Transfusion Reactions
  - Transfusion Associated Graft versus Host Disease (TA-GVHD)
  - Post-Transfusion Purpura (PTP)
  - Neonatal Alloimmune Thrombocytopenia (NAIT)
  - Iron Overload

Febrile, Nonhemolytic Transfusion Reactions

- General Information:
  - Relatively common
  - ~1:200 transfusions
  - No long-term impact on the patient
- Recognition:
  - 1 – 2° ↑ temperature during or within 4 hours of transfusion
  - ± chill / rigors
  - *Often very difficult to distinguish between febrile transfusion reaction and a fever related to the patient's underlying condition*
- Pathophysiology:
  - White blood cells and cytokines (IL-1, IL-6, IL-8, TNFα) elaborated in the donor blood product
- Treatment / Prevention:
  - Antipyretics
  - ***Leukoreduction*** of blood products reduces (but does not eliminate) the risk of febrile reactions

TA-Graft-vs.-Host Disease

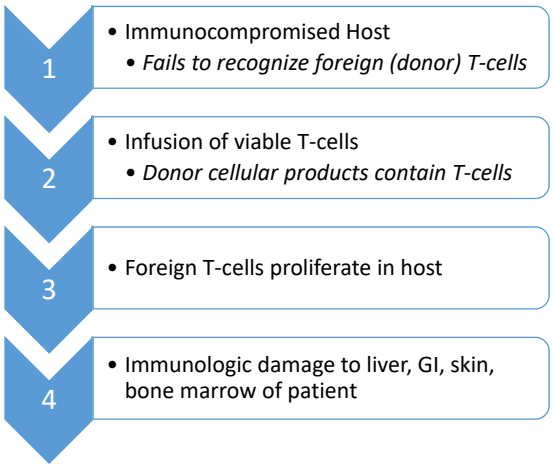
- General Information:
  - Very rare
  - Generally affects ***only*** severely immunocompromised patients such as those who have:
    - Congenital immunodeficiencies
    - Hodgkin's disease
    - Intrauterine transfusions
    - Low-birthweight neonates
    - Bone marrow transplant patients
    - Recipients of HLA-matched blood products
- Recognition:
  - Rash



  - Liver function test abnormalities
  - Diarrhea
  - Nausea/vomiting
  - Pancytopenia
  - Symptoms begin 7-10 days post-transfusion

TA-Graft versus Host Disease

- Pathophysiology:
  - In patients with a "normal" immune system, viable lymphocytes found in any cellular blood product (*WB, RBCs, Plts, Granulocytes*) would be recognized and destroyed.
  - In severely immunocompromised patients, this recognition of donor (foreign) lymphocytes does not occur allowing the donor lymphocytes to "colonize" the patient and proliferate
  - GvHD is the immunologic manifestation of tissue damage as the result of the proliferation of donor lymphocytes in an immunocompromised host



TA-Graft versus Host Disease

- Treatment:
  - Immunosuppressive therapies used in transplant-associated GvHD are generally **ineffective** in *Transfusion Associated GvHD (*TA-GvHD)
  - Most patients (>90%) die within 1 – 3 weeks of the onset of symptoms
- Prevention:
  - The only effective method for the prevention of GvHD is ***irradiation*** of all cellular blood products

Post-Transfusion Purpura (PTP)
Neonatal Alloimmune Thrombocytopenia (NAIT)

- In some ways these two transfusion reactions are very similar and in other ways quite different:

Similarities

- Both are associated with severe thrombocytopenia (Significant bleeding risk)
- Both have characteristic symptoms of ↓platelet count (petechiae, bruising, mucosal bleeding)
- Both are caused by the development of alloantibodies to **platelet-specific antigens** (particularly HPA-1a *aka* GPIIIa) *(~2% of the population lacks HPA-1a and can make an antibody to it)*

Differences	
Post-Transfusion Purpura	**Neonatal Alloimmune Thrombocytopenia**
• Adults • Sudden onset of thrombocytopenia 5-10 days after a transfusion • Platelet count <10,000 • Patient has a previous history of transfusion or pregnancy	• Fetal / Neonatal • Platelet count <20,000

Post-Transfusion Purpura (PTP)
Neonatal Alloimmune Thrombocytopenia (NAIT)

- **Posttransfusion Purpura (PTP):**
  - In patients who are negative for the HPA-1a antigen, platelet or red cell transfusion can lead to the development of a potent anti-HPA-1a IgG antibody
  - Usually this represents an anamnestic antibody response in a patient who was previously sensitized by a prior transfusion or during pregnancy
  - Uniquely, in PTP not only are HPA-1a transfused platelets destroyed by the patient's anti-HPA-1a antibody, but the patient's own HPA-1a NEGATIVE platelets are destroyed in an "innocent bystander" reaction (probably due to the formation of immune complexes)
  - PTP can result in severe thrombocytopenia
- **Neonatal Alloimmune Thrombocytopenia (NAIT):**
  - Women negative for HPA-1a antigen can develop an anti-HPA-1a, IgG antibody during pregnancy (when fetus is HPA-1a positive—and most are!!!)
  - This antibody can cross the placenta and, if the fetus is HPA-1a antigen positive, can sensitize the fetal platelets and ultimately result in severe thrombocytopenia in the fetus
  - NAIT can be manifest in the first pregnancy
  - When one infant is affected by NAIT there is a very high probability that subsequent fetuses will be affected

Post-Transfusion Purpura (PTP)
Neonatal Alloimmune Thrombocytopenia (NAIT)

- **PTP:**
  - Treatment
    - IVIg
    - HPA-1a NEGATIVE platelets can be helpful in the acute phase (*BUT, these transfused platelets have a much reduced survival*)
    - Future transfusions should utilize red cells and platelets known to be HPA-1a NEGATIVE
- **NAIT:**
  - Treatment
    - Antenatal IVIg with or without steroids
    - Intrauterine platelet transfusion using HPA-1a NEGATIVE platelets

Iron Overload

- Iron Overload is a complication that almost exclusively affects patients who are chronically transfused in the absence of bleeding
  - The patients most commonly affected are those with sickle cell disease or thalassemia
  - Each RBC unit contains ~200-250 mg iron; maximum daily excretion of iron ~1-2 mg
  - As transfused RBC become senescent or are destroyed, the liberated iron cannot be effectively eliminated and begins to accumulate in liver, heart, pancreas, kidneys and other organs
  - A cumulative dose of 50-100 RBC units can result in significant morbidity and mortality

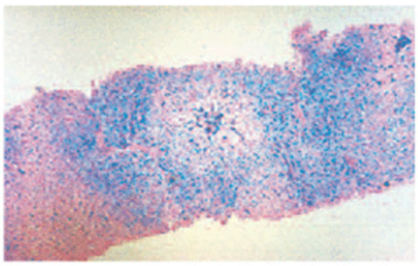


Figure 2—Iron deposition. Prussian blue iron stain of liver tissue (4x).

- **Symptoms:** lethargy, fatigue; liver damage, increased risk of diabetes, heart disease, arthritis
- **Treatment:** chelation therapy (deferoxamine, deferipone, or deferasirox)

Transfusion-Transmitted Infections (TTI)
(Infectious Complications)

- A wide variety of infectious agents can be transmitted by blood transfusion:

	VIRUS	BACTERIA	PROTOZOA	OTHER
Current TTI	Hepatitis B Hepatitis C HIV HTLV West Nile Virus Cytomegalovirus (CMV) Hepatitis A	Trepanema pallidum (syphilis) Bacteria as blood component contaminants	Plasmodium sp. (Malaria) Trypanosoma cruzi (Chagas) Babesia sp	Prions (variant CJD)
Probable TTI	Dengue HHV-8 Parvovirus B19 Chikungunya virus	Borrelia (Lyme dis.) Rickettsia	Leishmania sp.	

- But in spite of the variety of agents, the overall risk of transfusion-transmitted infection (TTI) is very small due to enhanced ***testing*** and ***donor screening***.

Transfusion-Transmitted Infections (TTI)
(Infectious Complications)

- Reducing the risks of TTI has been very successful and has utilized a variety of approaches:
  - Questioning the potential donor about certain risks
  - Testing donated blood for specific infectious agents
  - Geographic deferrals
- The current estimated risks of certain infectious agents are:

(NAT = Nucleic Acid Test; verys sensitive and specific (similar to PCR)

Infectious Agent	Testing	Residual Risk/Unit
HIV 1,2	Anti-HIV; NAT + Questions	1:2,135,000
HCV	Anti-HCV; NAT + Questions	1:1,935,000
HAV	Questions; no tests	1:1,000,000
HBV	HBsAg; NAT; Questions	1:400,000
HTLV I/II	Anti-HTLV	1:2,993,000
Parvovirus B19	None	1:40,000
Trypanosoma cruzi	EIA	Unknown

Infectious Complications—Bacterial Diseases

- The transmission of bacteria through blood transfusion is a mix of possible risks:
  - *Treponema pallidum*
    - A test for syphilis was the first "infectious disease" test used in screening blood products, but is of little practical benefit in enhancing blood transfusion safety
  - Bacterial contamination of blood products
    - Accidental contamination of blood products, usually at the time of donation, represents a real and significant threat to the safety of transfusion

Bacterial Contamination of Blood Products

- Blood products can become contaminated in 3 possible ways:
  - Bacteria from the donor's skin
  - Donor experiencing bacteremia
  - Blood collection bag is contaminated at manufacture (EXTREMELY RATE)
- The risk of bacterial contamination is 10-20 times greater with platelet units as compared to red blood cell units; probably due to the fact that platelets are stored at 20-24^{o} C while red blood cells are stored at 1-6^{o} C
- Due to the increased risk of bacterial contamination of platelet products, all platelets are screened for bacteria 24 hours after collection (before they are transfused) and then again at day 3-4
- Many types of bacteria have been implicated, but the most common are:
  - Red blood Cells:
    - *Yersinia enterocolitica*
    - *Pseudomonas*
  - Platelets:
    - *Staphylococcus*
    - Miscellaneous skin flora

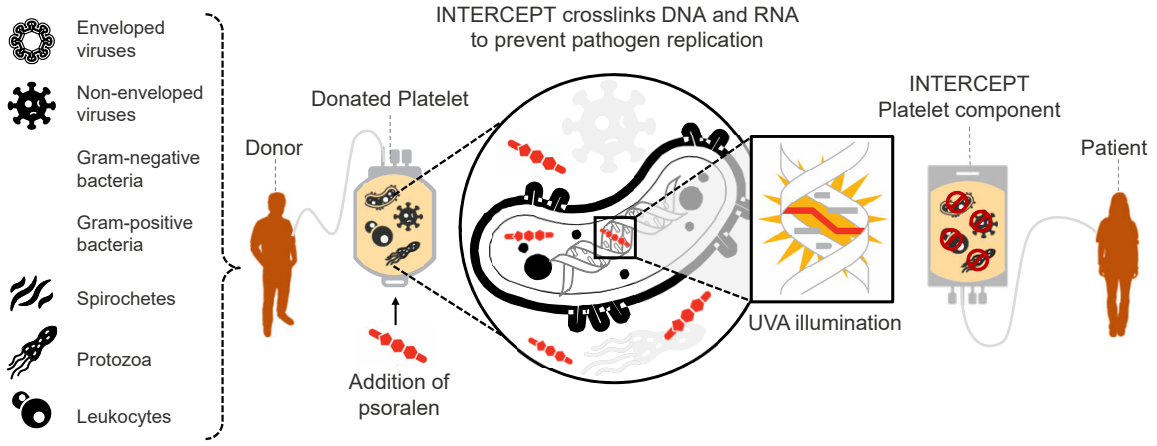
Bacterial Contamination of Blood Products

- Symptoms:
  - Fever (often >38.5° C)
  - Nausea / vomiting
  - Dyspnea
  - Hypotension
  - Diarrhea
  - DIC
  - **Mortality rate up to 70%**

Pathogen Reduction

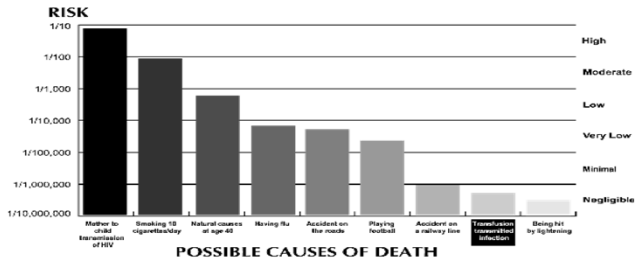
Platelets and Cryoprecipitate

- Pathogen Reduction
  - Recently, new technologies have become available in the U.S. that can be used to further reduce the risk of Transfusion Transmitted Infections (TTI)
    - The currently available technology uses a psoralen reagent that is added to platelet or cryoprecipitate products
    - The products are then irradiated with ultraviolet light which causes the psoralen reagent to covalently bind to the DNA or RNA of viruses, bacteria, parasites, and lymphocytes preventing replication
    - This treatment essentially renders these products nearly “risk-free” with respect to TTI
- The next slide diagrammatically outlines the Pathogen-Reduction process……

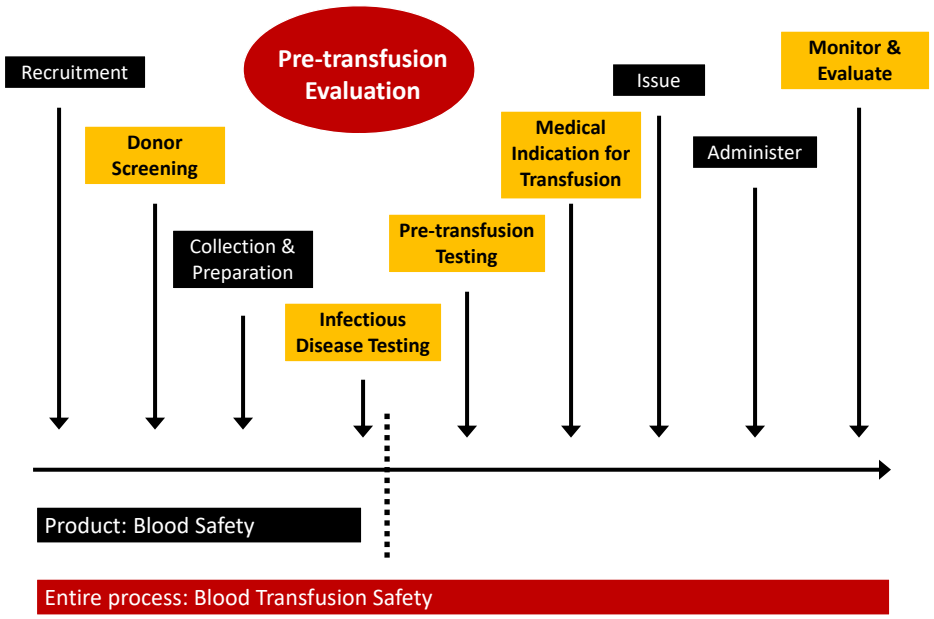


The Safety of Blood Transfusion--Summary

- Overall blood transfusion is remarkably safe so far as medical therapies go.
- Comparing transfusion to other tangible risks:



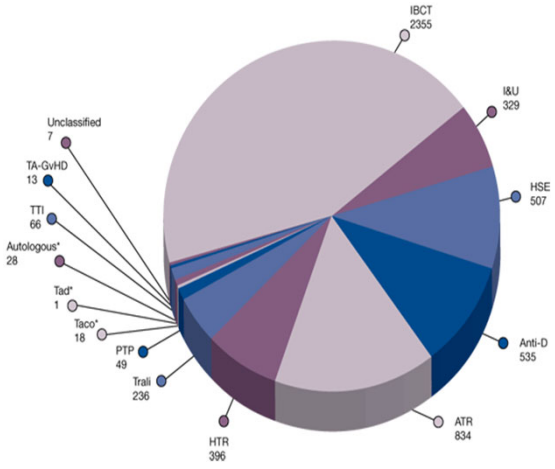
- ***Overall what accounts for this relatively low risk level?***



Transfusion Safety—A Final Note of Caution!

- The ***real*** risk of blood transfusion as revealed by hemovigilance data:
  - TTI: 1.2%
  - GvHD: 0.2%
  - TRALI: 4.5%
  - Febrile and Allergic: 15.5%

- **ERROR: 43.8%**



Practice Exercises:

- It is important to be able to recognize the signs and symptoms each of the classic transfusion reactions; complete the table on the next slide listing the major signs and symptoms of each transfusion reaction and the basics of therapy for each.

Transfusion Reactions

Summary of Symptoms

	Allergic	Anaphylactic	Febrile	TRALI	TACO	Acute Hemolytic	Delayed Hemolytic	Bacterial Contam.
Symptoms								
Treatment								

Don't forget to review: *PTP, NAIT, Iron Overload, and GvHD*

Answers:

	Allergic	Anaphylactic	Febrile	TRALI	TACO	Acute Hemolytic	Delayed Hemolytic	Bacterial Contam.
Symptoms	Urticaria Pruritis Flushing Angioedema Wheezing	Urticaria Pruritis Flushing Angioedema Wheezing Hypotension (severe)	Fever (↑1-2° C) Chills Rigors	Dyspnea Pul. Infiltrate (on CXR) Fever Chills Hypotension CVP (normal to low)	Dyspnea Pul. Infiltrate (on CXR) Orthopnea Hypertension CVP (elevated)	Fever Chills Dyspnea Pain Hypotension Hgb in blood and urine ↓Haptoglobin ↑LDH Jaundice	Minimal symptoms Anemia (unexpected) Jaundice	Fever Nausea Vomiting Dyspnea Hypotension Diarrhea DIC
Treatment	Suspend Txf Antihist.	Stop Txf Antihist. Stabilize BP	Stop Txf Leukoreduction Tylenol	Stop Txf Resp. support Stabilize BP	Stop Txf Diuretics	Stop Txf Maintain urine output Stabilize BP Monitor Coags	Often no therapy req.	Maintain BP Monitor Coag Antibiotics