[Some] Adverse Effects of Blood Transfusion

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Kermanshah blood transfusion conference - 4/feb/2019
Blood Transfusion is “Unavoidably Unsafe”

• High volume
• High cost
• High Risk
• Problem prone
The Risk Side of the Transfusion Equation

- Blood transfusions are the *most common procedure* performed on inpatients in the US.
- Over **21 million** blood components are transfused annually in the US to more than 5 million patients.
- Blood transfusions remain the #1 most common *allogeneic tissue transplant* in medicine.
- Blood transfusions expose patients to allogeneic antigens much more commonly than the sum of all other tissues and organs transplanted worldwide.
The Risk Side of the Transfusion Equation

• Infectious disease risks have been vastly reduced over the last 35 years
  ➢ The viral risks remain “non-zero”
  ➢ Bacteria are the most common pathogens
  ➢ Constant concerns for emerging pathogens

• Acute adverse effects remain common
  ➢ Irreducible human errors
  ➢ Irreducible immunologic risks due to use of allogeneic tissue donors
There are 35 Blood Group Systems & ~ 350 authenticated Blood Group Antigens
HLA Antigens – Expressed on WBC’s - are
- **THE** Tissue-Typing Antigens
- Are the most polymorphic in man
- Basis of Immune-mediated platelet refractoriness

**HLA class I and class II antigens**

- Monomer with non-covalently associated subunit (β2m)
- Presents antigenic peptides to CD8+ T cells
- Expressed by all nucleated cells

- Heterodimer
- Presents antigenic peptides to CD4+ T cells
- Restricted expression on antigen presenting cells (dendritic cells, B cells, macrophages)
- Inducible on other cells (endothelium and epithelium)
Platelet-Specific Alloantigens

• 24 platelet-specific alloantigens defined by immune anti-sera
• 16 are grouped into 8 bi-allelic systems
• The alleles differ by a SNP
• The molecular basis of 22 of 24 have been resolved
• Platelet-specific antibodies are the pathophysiological basis of
  ➢ Posttransfusion purpura (PTP)
  ➢ Neonatal alloimmune thrombocytopenia (NAIT)
Platelet-Specific Alloantigen Families: “Human Platelet Antigens” [HPA’s]

<table>
<thead>
<tr>
<th>Antigen Family, Allelic Pairs</th>
<th>Epitopes on Specific Membrane Glycoproteins</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPA-1a/1b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-2a/2b</td>
<td>GP-Ib-IX</td>
</tr>
<tr>
<td>HPA-3a/3b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-4a/4b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-5a/5b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-6a/6b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-9a/9b</td>
<td>GP IIb/IIIa</td>
</tr>
<tr>
<td>HPA-15a/15b</td>
<td>CD 109</td>
</tr>
</tbody>
</table>
## RESIDUAL INFECTIOUS DISEASE RISKS in TRANSFUSION

<table>
<thead>
<tr>
<th>Disease Transmitted by Blood</th>
<th>Estimated Frequency per Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B virus</td>
<td>1 : 843,000 – 1,280,000</td>
</tr>
<tr>
<td>Hepatitis C virus</td>
<td>1 : 1,149,000</td>
</tr>
<tr>
<td>HIV- 1 / 2</td>
<td>1 : 1,470,000</td>
</tr>
<tr>
<td>HTLV – I / II</td>
<td>&lt; 1 : ~ 3,000,000</td>
</tr>
<tr>
<td>WNV</td>
<td>&lt;&lt; 1 : ~ 4,000,000</td>
</tr>
<tr>
<td>Bacterial contamination (platelets)</td>
<td>1 : ~ 2000 – 3000 platelet transfusions</td>
</tr>
</tbody>
</table>
Next *Diseases of Concern for Testing in the Blood Supply*

- Arboviruses
  - Zika, Dengue, Chikungunya
- *Babesia* species
- Parvovirus B19
- Human v-CJD

_Emerging Infectious Diseases and their potential threat to transfusion safety, Transfusion* 2009;49, 1S-29S._
Critical & Fundamental Process Defects

• Blood transfusion is “unavoidably unsafe”
• Transfusions = allogeneic tissue transplants
• The complex series of processes to deliver transfusions are performed by imperfect humans
• Process control is often lacking
Transfusion Therapy is a Set of Processes, not just a *Product* or a *Lab Result*

**Product:** Blood safety

**Entire process:** Safe Blood Transfusion
Systematic Review of RBC Transfusion in the Critically Ill

• 45 studies with a median of 687 patients per study

• Outcome measures, attributed to “immunomodulation”
  ➢ Mortality
  ➢ Infection
  ➢ MOF Syndrome
  ➢ ARDS

• Risks of RBC transfusion outweighed benefits in 42 of 45 studies!!

Association of Transfusion & Risk of Death

Figure 2. Association between blood transfusion and the risk of death (odds ratio [OR] and 95% confidence interval [CI]). ACS, abdominal compartment syndrome; ICU, intensive care unit.

Transfusion Reactions

- Acute (intravascular) hemolytic reaction
- Delayed (extravascular) hemolytic reaction
- Febrile non-hemolytic reaction
- Allergic (urticarial) reaction
- Bacterial contamination
- Transfusion-related acute lung injury
- Transfusion-associated circulatory overload
- Post-transfusion purpura
- Graft-vs.-host disease
Acute Adverse Effects

• Immune-Mediated
  - Acute hemolytic TR’s
  - Fever without hemolysis: FNHTR’s
  - Simple *allergic* reactions
  - TRALI
  - Anaphylactoid / anaphylactic reactions
Acute Adverse Effects

• **Nonimmunologic**

  - Transfusional hypervolemia (circulatory overload), *aka TACO*
  - Bacterial septic reaction
  - Isolated Hypotensive TR’s
  - Citrate toxicity
  - Nonimmune hemolysis (often asymptomatic)
Acute Transfusion Reactions by Systemic Manifestations

• **Fever and/or chills** (including rigors), no hemolysis
  - R/o acute, immune-mediated hemolysis
    - Work-up required by laboratory Standards
  - FNHTRs
  - Bacterial contamination

• **Allergic (Type I hypersensitivity reactions)**
  - Mucocutaneous (pruritus / urticaria) vs. more generalized, involving other organ systems

• **Respiratory Distress**
  - TACO
  - TRALI
  - May be part of anaphylactoid or anaphylaxis

• **Isolated Hypotensive TR’s**
  (without cardiovascular collapse)
# Estimated Frequencies of Some Adverse Effects of Transfusion

<table>
<thead>
<tr>
<th>TYPE of Reaction</th>
<th>Reported Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TACO</strong></td>
<td>1 : 68 to 1 : 356 (ICU patients)</td>
</tr>
<tr>
<td></td>
<td>[Mortality 1 to 3%]</td>
</tr>
<tr>
<td><strong>TRALI</strong></td>
<td>1 : 1200 to 1 : 190,000</td>
</tr>
<tr>
<td></td>
<td>[Leading reported cause of transfusion-related mortality]</td>
</tr>
<tr>
<td>WBC alloimmunization</td>
<td>1 : 20 to 1 : 100</td>
</tr>
<tr>
<td>FNHTR’s</td>
<td>1 : 100 – 200</td>
</tr>
<tr>
<td>Cutaneous allergic</td>
<td>1 : 100 – 300</td>
</tr>
<tr>
<td>Delayed serologic</td>
<td>1 : 1500</td>
</tr>
<tr>
<td>Delayed hemolytic</td>
<td>1 : 4000</td>
</tr>
<tr>
<td>Anaphylactoid</td>
<td>1 : 20,000</td>
</tr>
<tr>
<td>Acute hemolysis</td>
<td>1 : 6,000 – 33,000</td>
</tr>
<tr>
<td>Anaphylactic</td>
<td>1 : 20,000 to 1 : 50,000</td>
</tr>
<tr>
<td>Hypotensive TR’s</td>
<td>1 : 18,500</td>
</tr>
</tbody>
</table>
Preventing Acute Hemolysis due to Pre-formed IgM, as seen in ABO-incompatibility

• All steps in **specimen** & **patient** identification are aimed at prevention!

• All pretransfusion testing is done to prevent acute intravascular hemolysis
  - Blood typing & confirmation
  - Antibody screening
  - Crossmatching
Acute Complement-Mediated, Immediate Hemolysis

The Direct Antiglobulin Test will be positive
Acute Intravascular Hemolytic Transfusion Reaction

IMMEDIATE HEMOLYTIC REACTION

SYMPTOMS
- FEVER
- CONSTRICION OF CHEST
- PAIN IN LUMBAR REGION

SIGNS
- FEVER
- HYPOTENSION
- HEMOGLOBINURIA
- BLEEDING
- RENAL FAILURE

Main symptoms of Acute hemolytic reaction

Systemic
- Chills
- Fever

Vascular
- Hypotension
- Uncontrollable bleeding

Transfused vein
- Heat sensation

Chest
- Constricting pain

Urinary
- Hemo-
- globinuria
- Hyper-
- bilirubin-
- emia

Heart
- Increased heart rate

Lumbar region
- Pain
Freshly Hemolyzed Plasma (Actual Case from an OR)

Dr. DeChristopher’s gloved fingers
Febrile Nonhemolytic Transfusion Reactions

- **Cardinal Signs & Symptoms** (near end of transfusion or up to 2 hours posttransfusion)
  - Fever
  - Chills / *cold feeling* are more common than frank *fever*
  - General discomfort
  - Less commonly rigors, nausea/vomiting, dyspnea
  - Not associated with clinical hemolysis (by laboratory testing)
FNTRs

• **Common Etiologies**

  ➢ Pre-formed recipient WBC antibodies
    o Chronically, heavily transfused recipients
    o Multi-gravid females
    o Solid-organ transplant recipients
  ➢ *Storage lesion* cytokines, termed a “Cytokine Storm”
    o Proinflammatory cytokines, such as IL-1, IL-2, IL-6, IL-8, TNF-\(\alpha\), in the blood components
Febrile Reactions: Rule Out Hemolysis & Acute Bacterial Sepsis
Bacterial Contamination and/or Endotoxemia

Diagram showing SEPTICEMIA with symptoms:
- Fever
- Chills
- Hypotension
- Death
The Cavalcade of Bacterial *Stars* Implicated in Transfusion-transmitted Septic Deaths

- *Staphylococcus aureus*: 17%
- *Staphylococcus epidermidis*: 10%
- *Klebsiella pneumoniae*: 17%
- *Escherichia coli*: 6%
- *Serratia marcescens*: 16%
- *Salmonella species*: 8%
- *Enterobacter cloacae*: 6%
- *Pseudomonas aeruginosa*: 6%
- *Group B streptococcus*: 8%
- *Bacillus cereus*: 8%
Simple Cutaneous Hypersensitivity

- Usually involves the skin and is limited anatomically
- Common, but usually mild / self-limited
- More common in recipients of large volumes of plasma
- The transfusion can temporarily be halted
- Consider administration of antihistaminic medications
- Can restart the transfusion if non-progressive
- Not required by AABB Standards to be reported as a “transfusion reaction”
Allergic Transfusion Reactions: Hives and Itching
Acute Anaphylactic Shock

- Acute cardiopulmonary collapse
- Evolves RAPIDLY
- Plasma proteins are etiologic
- Cannot be predicted or prevented
- “Reactions” occur after exposure to only small quantities of blood
- Will likely require ACLS
Consensus Definition of TRALI

- A new ALI within 6 hours of a completed transfusion
- No other temporally-associated ALI risk factors
- TRALI is a *clinical syndrome* rather than a disease with a single etiology
- It is a clinical and radiographic diagnosis
- TRALI is NOT diagnosed based on laboratory test results
• Acute onset
• Hypoxemia
  ➢ Research setting
    o $\text{PaO}_2 / \text{FiO}_2$ ratio $\leq$ 300mm Hg or
    o $\text{SpO}_2 < 90\%$ on room air
  o Non-research setting
    o As above or other clinical evidence of hypoxemia
• Bilateral infiltrates on frontal CXR
• No evidence of left atrial hypertension (circulatory overload)

Transfusion-Related Acute Lung Injury (TRALI)

• Very common signs / symptoms
  – Dyspnea, respiratory distress, hypoxia, bilateral pulmonary edema, fever (1 – 2 degree increase)

• Common signs / symptoms
  – Tachycardia, hypotension, cyanosis

• Diagnosis includes ruling out cardiogenic causes of pulmonary edema

• A clinical diagnosis of exclusion
1\textsuperscript{st} Basic Mechanism Proposed for TRALI Pathogenesis

• 1\textsuperscript{st} Mechanism (antibody-mediated):
  ➢ Specific antibodies
    o Usually HLA Class I or anti-granulocyte
  ➢ PMN’s with cognate antigens
  ➢ Pulmonary leukostasis and PMN activation
  ➢ (Positive complement activation)
  ➢ Endothelial injury, capillary leak, ALI
2nd Basic Mechanism Proposed

• *Two-Hit*, two independent events:
  - **Systemic inflammation** (patient *primed* by underlying clinical condition)
  - Release of *proinflammatory mediators*
    - Inflammatory cytokines
    - Storage lesion lysophosphatidylcholines & neutral lipids
  - Pulmonary leukostasis, PMN activation and release of “reactive oxygen species”
  - Endothelial injury, capillary leak, ALI
The Business End of Respiration and of TRALI
Diffuse Alveolar Damage

Hyaline Membranes
Pulmonary Congestion and Edema
# Transfusion Reactions with Respiratory Symptoms: TRALI

<table>
<thead>
<tr>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Higher odds among specific groups</td>
</tr>
<tr>
<td>- Age 65 – 79 vs. older than 79 years</td>
</tr>
<tr>
<td>- Rates higher for platelet and plasma-containing transfusions</td>
</tr>
<tr>
<td>- Females vs. males</td>
</tr>
<tr>
<td>- White vs. nonwhite</td>
</tr>
<tr>
<td>- (Incidence 0.02%)</td>
</tr>
<tr>
<td>• Post-inflammatory pulmonary fibrosis</td>
</tr>
<tr>
<td>• Cancers of blood forming tissues</td>
</tr>
<tr>
<td>• Pulmonary insufficiency following trauma or surgery</td>
</tr>
<tr>
<td>• Tobacco use</td>
</tr>
<tr>
<td>• Blood transfusion</td>
</tr>
</tbody>
</table>
Transfusional Hypervolemia, aka TACO
### Transfusion Reactions with Respiratory Symptoms: TACO

#### Risk Factors

- **Patient demographics**
  - Age (60% ≥ 70 years of age)
  - ≥ 80 years, **4-fold higher** rate (7.4% vs. 2.0%)
  - 1 : 68 – 1 : 1566 risk in **plasma recipients**
  - 1 : 356 transfused ICU patients

- **Medical Conditions (critically ill vulnerable)**
  - Chronic renal failure (OR **27.0**)
  - Left ventricular dysfunction (OR **8.23**)
  - Congestive heart failure (OR **6.6**)
  - Blood transfusion (OR **1.11** / unit)

- **Perioperative setting (4.3% incidence)**
  - Vascular, transplant & thoracic surgeries highest rates
  - Increasing transfusion volumes
  - Positive fluid balance
Transfusional Hypervolemia, aka TACO

• Must Be Distinguished from TRALI
• TACO Kills!!

• Some Distinguishing Characteristics
  - Usually affects patients at the age extremes
  - Evaluate fluid status (I’s & O’s)
  - Evaluate BNP
  - SOB, pulmonary congestion, distended neck veins, cyanosis, peripheral edema

• Preventive Measures
  - Avoid transfusions!
  - Slower transfusion rates
  - Aliquoting components
  - Concomitant diuresis / volume reduction
## Comparing TRALI & TACO

<table>
<thead>
<tr>
<th>Similar Features</th>
<th>TRALI</th>
<th>TACO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td>Diffuse bilateral infiltrates</td>
<td>Diffuse bilateral infiltrates</td>
</tr>
<tr>
<td>Respiratory Symptoms</td>
<td>Acute dyspnea</td>
<td>Acute dyspnea</td>
</tr>
<tr>
<td>Auscultation</td>
<td>Rales</td>
<td>Rales</td>
</tr>
</tbody>
</table>

**TRALI** = transfusion-related acute lung injury;  
**TACO** = transfusion-associated circulatory overload

Comparing TRALI & TACO

<table>
<thead>
<tr>
<th>Disparate Features</th>
<th>TRALI</th>
<th>TACO</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Temperature</td>
<td>Often elevated</td>
<td>Often unchanged</td>
</tr>
<tr>
<td>• Blood pressure</td>
<td>Hypotension</td>
<td>Hypertension</td>
</tr>
<tr>
<td>• Pulmonary artery occlusion pressure</td>
<td>≤ 18 mm Hg</td>
<td>&gt; 18 mm Hg</td>
</tr>
<tr>
<td>• Response to diuretic</td>
<td>Minimal</td>
<td>Significant</td>
</tr>
<tr>
<td>• WBC count</td>
<td>May have transient leukopenia</td>
<td>Unchanged</td>
</tr>
<tr>
<td>• Pulmonary edema fluid</td>
<td>Exudate</td>
<td>Transudate</td>
</tr>
<tr>
<td>• Fluid balance</td>
<td>Positive, even, negative</td>
<td>Positive</td>
</tr>
</tbody>
</table>

- Patients with either may lack *typical* features
- Patients with TRALI may have TACO features
- TRALI & TACI may present concurrently
## TRALI vs. TACO

<table>
<thead>
<tr>
<th>TRALI</th>
<th>TACO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signs &amp; Symptoms</strong></td>
<td><strong>Signs &amp; Symptoms</strong></td>
</tr>
<tr>
<td>Respiratory distress</td>
<td>Respiratory distress</td>
</tr>
<tr>
<td>Tachypnea</td>
<td>Tachypnea</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Hypotension</td>
</tr>
<tr>
<td><strong>Noncardiogenic</strong> pulmonary edema</td>
<td><strong>Cardiogenic</strong> pulmonary edema</td>
</tr>
<tr>
<td>Fever</td>
<td>Improves with diuretics</td>
</tr>
<tr>
<td>Onset within 6 hours of transfusion</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Supporting Data</strong></th>
<th><strong>Supporting Data</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>B/L pulmonary infiltrates on CXR</td>
<td>B/L pulmonary infiltrates on CXR</td>
</tr>
<tr>
<td>Decreased WBC count</td>
<td>Pretransfusion fluid overload</td>
</tr>
<tr>
<td><em>Associated with</em> HLA and/or Neutrophil Antibodies</td>
<td>Elevated BNP</td>
</tr>
<tr>
<td></td>
<td>Increased heart size</td>
</tr>
<tr>
<td></td>
<td>Vascular congestion</td>
</tr>
<tr>
<td></td>
<td>Pulmonary wedge P &gt; 18 mm Hg</td>
</tr>
</tbody>
</table>
Admission Parameters

• 77-y/o female with PMH of invasive ductal breast carcinoma and SLL / CLL, both 4 years PTA

• CBC showed: H & H of 5.8 g/dL & 17.4%; WBC 119 K / µL (98% lymphs); platelets 25 K / µL

• The CLL was “end-stage”, refractory to all prior treatment attempts

• Patient refused further oncologic therapy!!

• Admitted for evaluation and treatment of cytopenias, specifically the anemia

• Plan was to provide RBC transfusion, then d/c to home or other hospice care.
Admission Parameters

• Patient had a chronic UTI x 3 months, sinus congestion and a productive cough (no evidence of pneumonia), aFib with RVR

• Transfusion support (cellular components):
  - Irradiation
  - Leukoreduction
  - CMV-seronegative
  - XM-compatible

• Three units of RBC were ordered

• The patient was pre-medicated with 375 mg of acetaminophen
### Three RBC Transfusions Given over an Elapsed 10-Hour Period

**#1: Begun 00:45, Completed 03:35**

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Pre-Txn</th>
<th>Post-Txn</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temp (°C)</td>
<td>36.3</td>
<td>36.6</td>
</tr>
<tr>
<td>Pulse</td>
<td>66</td>
<td>80</td>
</tr>
<tr>
<td>BP</td>
<td>108/53</td>
<td>119/55</td>
</tr>
<tr>
<td>Respirations</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>

**#2: Begun 04:10, Completed 07:00**

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Pre-Txn</th>
<th>Post-Txn</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temp (°C)</td>
<td>36.6</td>
<td>36.5</td>
</tr>
<tr>
<td>Pulse</td>
<td>70</td>
<td>64</td>
</tr>
<tr>
<td>BP</td>
<td>108/54</td>
<td>144/80</td>
</tr>
<tr>
<td>Respirations</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>

**#3: Begun 08:00, Completed 10:00**

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Pre-Txn</th>
<th>Post-Txn</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temp (°C)</td>
<td>36.5</td>
<td>36.9</td>
</tr>
<tr>
<td>Pulse</td>
<td>64</td>
<td>87</td>
</tr>
<tr>
<td>BP</td>
<td>144/80</td>
<td>196/84</td>
</tr>
<tr>
<td>Respirations</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>
Unintended Consequences

• 12:00: Anxiety & agitation set in, but breathing normal
• 14:00: Dyspnea & increased WOB began
• 18:00: Cough developed with frothy, bloody sputum, AMS, lethargy, a/w desaturations → intubated & MICU transfer

• New-onset symptoms, associated abnormalities:
  - Positive fluid balance - Net I’s / O’s + 2600 mL
  - CXR with cardiomegaly new B/L lower lobe opacities
  - EKG with atrial flutter & new RBBB
  - Troponin I level at 0.69 ng/mL
  - BNP 743 pg/mL

• Worsening, refractory hypotension. DNR status & comfort care elected. Patient expired 12 hours posttransfusion.
And Your Diagnosis Is?

• Narrow Differential
  ➢ TACO vs. TRALI

• Reportabilities?
  ➢ To whom would YOU report this event?
    o The Blood Bank, order a “Transfusion Reaction Work-up”
    o Your immediate superiors

• To whom is such events required to be reported?
  ➢ Institutional Patient Safety / Risk Management
  ➢ Regional Blood Center (why?)
  ➢ The FDA!!

• One of 3 RBC donors was a female with broad HLA alloimmunization!! (Possible TRALI!!)
What’s Being Done to Mitigate Risks of Transfusion?

• Blood Center Donor Qualification, Unit Disease and other Testing:
  ➢ ABO, Rh, antibody screen
  ➢ Serologic tests
  o Syphilis (RPR or VDRL)
  o HBsAg, anti-HBc and anti-HCV
  o Retroviruses: Anti-HIV-1/2, anti-HTLV-I/II
  o Anti-*trypanosoma cruzi* (Chagas’ disease)

 ➢ Genomic amplification methods
  o HIV-1
  o HCV
  o HBV
  o WNV
  o Zika virus
TRALI Mitigation Strategies

• Provision & transfusion of “all-male” plasma
  - Successful in Europe since 2003
  - Difficult to meet AB plasma demands from male-only donors
  - The US incidence of FDA-reported TRALI deaths from plasma has significantly decreased since FY07
  - TRALI deaths still occur with RBC transfusion

• Qualify female platelet donors by testing for HLA and neutrophil antibodies
Other Risk Mitigation Strategies

• “Universal” leukoreduction
  • Reduces FNHTRs
  • Reduces alloimmunization to HLA antigens
  • Reduces platelet refractoriness
  • Reduces risk of CMV transmission
  • Reduces mediastinitis in CT surgery

• “All male” plasma selection (reduces TRALI risks)
• HLA / neutrophil Ab screening, female platelet donors
• Supplemental bacteriologic testing of platelets
• Repeat ABO / Rh testing to confirm recipient typing
• Selective recipients protections (e.g. CMV, irradiation)
• Application of pathogen inactivation techniques / products
What are **YOU** Prepared to Do to Mitigate Risks of Transfusion?

- Employ robust specimen & patient identification know-how
- *Discover the literature:* IDENTIFY the evolving evidence base & indications for transfusion therapy!
- Informed consent for transfusion: Describe & weigh risks!
- Limit or avoid transfusion (one element of *Patient Blood Management*)
  - Don’t order 2 when 1 will do!
- Learn how to appropriately order blood in Epic
- Recognize requirements for surgical blood ordering
- Report *suspected* Transfusion Reactions to Blood Bank
- Identify & use our institutional urgent agent reversal strategies in life-threatening bleeding
- *The Pharmacy is your friend!* (Alternatives to transfusion)
8 Rights of Transfusion Administration

8 RIGHTS:
- Product
- Patient
- Dose
- Time
- Reason
- Site
- Documentation
- Response
Transfusion Complications that Kill

- Acute intravascular hemolysis
- TACO
- TRALI
- Bacterial contamination
- Other microorganism contamination
- Anaphylaxis
- (Hyperhemolytic syndrome)
- Complications of Immunomodulation
- Acute TR graft-vs-host disease
The Risk Side of the Transfusion Equation

• Blood transfusion is a “liquid transplant”
• Blood transfusion risks can be mitigated, but not eliminated (“unavoidably unsafe”)
• There is always another “microorganism of the month” waiting in the wings
• Blood transfusions are the only tissues casually transplanted with the stroke of a mouse click
• The safest transfusion is the one you don’t give