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TRANSFUSION MEDICINE

- Focuses on the administration of blood, blood components, and purified blood proteins to patients for therapeutic purposes
- Hematopoietic progenitor cells (HPCs) are the most recently blood derived component to be developed for transfusion

DONATING BLOOD

A blood donor needs to:

be aged 16-70 years

Wt at least 45 kg

Be in good health, including Nl Temp, Bp.

Meet guidelines designed to protect the donor and people who will receive their blood.

TYPE OF BLOOD COLLECTION

Homologous

Aphaeresis

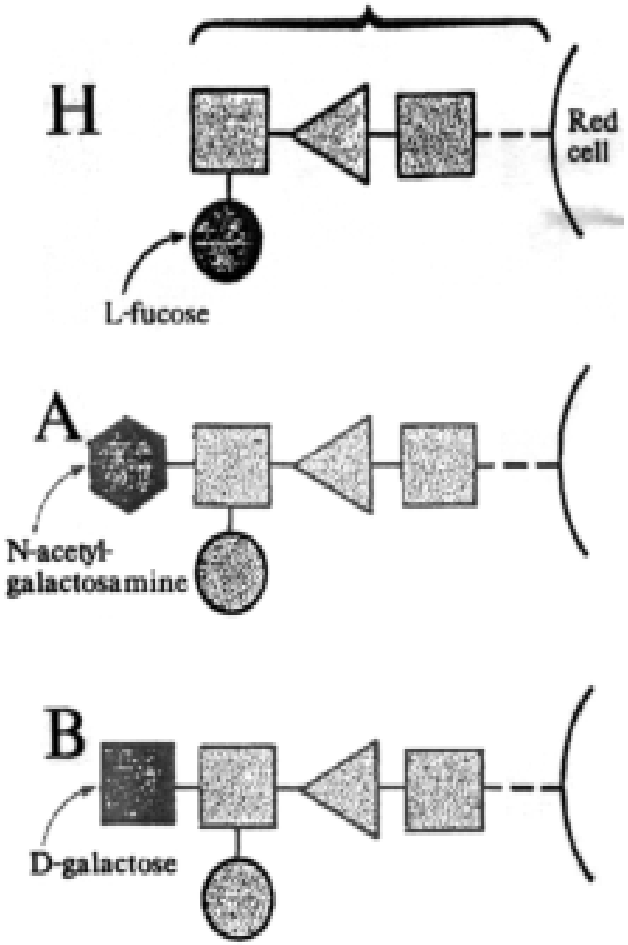
Autologous (reduces the possible risks of infections and incompatibility)

ABO BASICS

- Blood group antigens are actually sugars attached to the red blood cell.
- Antigens are “built” onto the red cell.
- Individuals inherit a gene which codes for specific sugar(s) to be added to the red cell.
- The type of sugar added determines the blood group.

THIS DIAGRAM ILLUSTRATES THE TERMINAL SUGAR FOR EACH BLOOD GROUP.

Schematic illustration of the oligosaccharide structures of the H, A and B antigens. The precursor chain is the substrate for the H gene-specified fucosyl transferase; the H chain is the substrate for both A and B gene-specified transferases, which add N-acetylgalactosamine and D-galactose, respectively, to the H chain. In the AB individual N-acetylgalactosamine and D-galactose are added to different chains of the same red cell. The symbols represent the following sugars: hexagon – N-acetylgalactosamine; square – D-galactose; triangle – N-acetylglucosamine; circle - L-fucose. The broken line represents additional sugars.



ABO TYPE FREQUENCIES

ABO Type	Per Cent
O	45%
A	40%
B	11%
AB	4%

LANDSTEINER'S RULE

- Individual's will form immune antibodies to ABO blood group antigens they do not possess.
- Substances are present in nature which are so similar to blood group antigens which result in the constant production of antibodies to blood group antigens they do not possess.
- Critical for understanding compatibility between ABO blood groups.

ANTIBODY CLINICAL SIGNIFICANCE

- Immunizations are frequently done to protect us from disease.
  - Receive Hepatitis B immunization.
  - Actual bits of hepatitis virus injected.
  - Body recognizes as foreign and produces an immune antibody.
  - Subsequent exposure to real Hepatitis B virus will result in destruction of the virus by immune antibodies.
- ABO antibodies are immune and will result in destroying incompatible cells which may result in the death of the recipient.

INHERITANCE

- Blood group antigens are “codominant”, if the gene is inherited, it will be expressed.
- Some aberrant genotypes do occur but due to the rarity will not be discussed.
- Understanding of basic inheritance important.

GENETICS

- Two genes inherited, one from each parent.
- Individual who is A or B may be homozygous or heterozygous for the antigen.
- Heterozygous: AO or BO
- Homozygous: AA or BB
- Phenotype is the actual expression of the genotype, ie, group A
- Genotype are the actual inherited genes which can only be determined by family studies, ie, AO.

EXAMPLE OF DETERMINING GENOTYPE

- Mom's phenotype is group A, genotype AO
- Dad's phenotype is group B, genotype BO

	B	O
A	AB 25%	AO 25% (Group A)
O	BO 25% (Group B)	OO 25% (Group O)

OTHER EXAMPLES

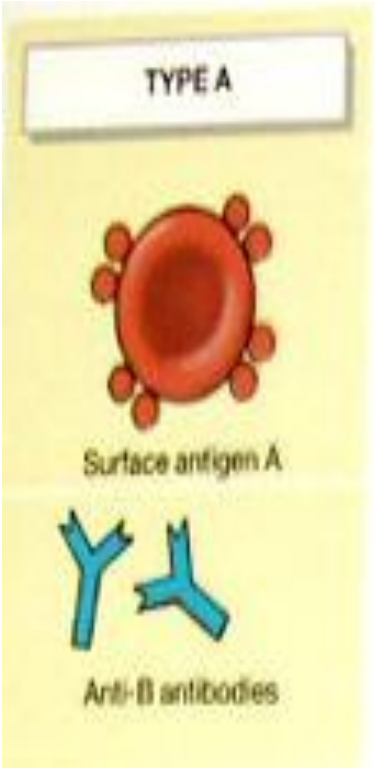
Mom	Dad	Offspring Blood Group
AA	BB	100% AB
BO	OO	50% each of B or O
OO	OO	100% O
OO	AO	50% each of A or O

GROUP O

- Approximately 45% of the population is group O.
- No A or B antigens present, think of as “0” antigens present.
- These individuals form potent anti-A and anti-B antibodies which circulate in the blood plasma at all times.

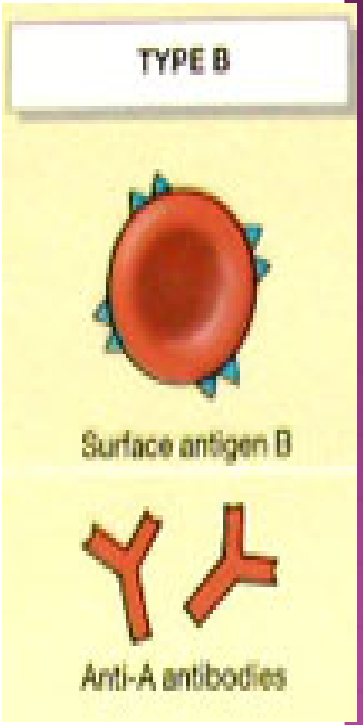
GROUP A

- Approximately 40% of the population is group A.
- No B antigens present.
- These individuals form potent anti-B antibodies which circulate in the blood plasma at all times.



GROUP B

- Approximately 11% of the population is group B.
- No A antigens present.
- These individuals form potent anti-A antibodies which circulate in the blood plasma at all times.



GROUP AB

- Approximately 4% of the population is group AB.
- Both A and B antigens present.
- These individuals possess no ABO antibodies.
- NOTE: This slide is in error as it only illustrates presence of one antigen not 2.

SUMMARY

Blood Group	Antigens on cell	Antibodies in plasma	Transfuse with group
A	A	Anti-B	A or O
B	B	Anti-A	B or O
AB	A and B	none	AB, A, B or O
O	None	Anti-A & B	O

Rh (D) Antigen

- Of next importance is the Rh type.
  - Term “Rh” is a misnomer.
  - Rh is a blood group system with many antigens, one of which is D.
  - Re-education of public is difficult.
- Rh refers to the presence or absence of the D antigen on the red blood cell.

RH (D) ANTIGEN (CONTINUED)

- Unlike the ABO blood group system, individuals who lack the D antigen do not naturally make it.
- Production of antibody to D requires exposure to the antigen.
- The D antigen is very immunogenic, ie, individuals exposed to it will very likely make an antibody to it.
- For this reason all individuals are typed for D, if negative must receive Rh (D) negative blood.

RH (D) ANTIGEN (CONTINUED)

- The most important patient population to consider is females of child-bearing age.
- If immunized to Rh (D) antigen the antibody can cross the placenta and destroy Rh (D) positive fetal cells resulting in death.
- This is why Rh negative women are given Rhogam after birth of Rh positive baby.

PACKED RED BLOOD CELLS

- The component of choice for replacement during surgery, red blood loss, and chronic transfusion therapy
- The Hct of the cells varies with the preservative solution used by the collection facility(approximately 75%)

- Collected in CPDA-1
- Red cells stored in Adsol or Nutricel have HCt 50-60%
- Adsol has manitol
- The effect of manitol?

BLOOD STORAGE

- Continue red cell metabolism and some leakage from RBC membrane: decreasing plasma dextrose & increase K
- Fresh blood: <7-10 days indicated in small patients receiving large amounts of blood

PRE TRANSFUSION TESTING AND COMPATIBILITY CONSIDERATIONS

A. Patients < 4 Months of Age

1. Tested for ABO and Rh type (cord blood or heel stick specimen).
2. An initial antibody screen must be done using a sample from either the infant or mother. If no unexpected antibodies are detected initially, the red blood cell (RBC) unit should be ABO compatible with the infant and mother and either Rh negative or of the same Rh group as the infant:
   a. Repeat ABO/Rh grouping is not required;
   b. Repeat antibody screening is not required;
   c. Compatibility testing is not required.

- Compatibility testing is required only under the following conditions (the mother's serum may be used):
- The infant has an unexplained positive direct coombs test result (e.g., not due to Rh immune globulin);
- An unexpected antibody is detected in the infant's or mother's serum or, alternatively, antigen-negative blood may be used without compatibility testing.

PATIENTS ≥ 4 MONTHS OF AGE

- 1. pretransfusion specimen for ABO/Rh and screened for unexpected antibodies.
- If no unexpected antibodies are detected, a crossmatch (if the blood group has been verified by a repeat sample or from prior history) may be performed with an ABO and Rh-compatible RBC unit.
- 2. Compatibility testing, including indirect antiglobulin phase, and selection of antigen negative RBCs (if applicable), are necessary under the following conditions:
  - an unexpected antibody is detected in the patient's serum;
  - the patient has a positive DAT test result;
  - the patient has a history of an antibody, not detectable currently.

GUIDELINES FOR TRANSFUSION OF RBCS IN PATIENTS < 4 MONTHS OF AGE

- Hb < 7 g/dL with low reticulocyte count and signs of anemia
- Acute blood loss of ≥ 15-20% of total blood volume
- Intra-operative blood loss of ≥ 15% of total blood volume
- Hb < 10 g/dL
  - CPAP/MV with mean airway pressure < 6 cm H2O
  - FIO2 < 35% via oxygen hood
  - On oxygen by nasal cannula
  - Significant apnea or bradycardia
  - Significant tachypnea or tachycardia
  - Low weight gain (100 kcal/kg/day)

Hb < 12 g/dL

- CPAP/MV with mean airway pressure > 6-8 cm H2O
- FIO2> 35% via oxygen hood

Hb < 15 g/dL with severe pulmonary or cyanotic heart disease/congestive heart failure or on ECMO

GUIDELINES FOR TRANSFUSION OF RBCS IN PATIENTS ≥ 4 MONTHS OF AGE

- Hb < 7 g/dL with low reticulocyte count and signs of anemia
- Acute blood loss of ≥ 15-20% of total blood volume
- Intra-operative blood loss of ≥ 15% of total blood volume
- Hb < 8 g/dL (Hct < 24%)

in the peri-operative period, with signs and symptoms of anemia

in congenital or acquired symptomatic anemia

in bone marrow failure

Hb < 13 g/dL with severe cardiopulmonary disease or on ECMO

Other inherited disorders of red cell production that require chronic transfusion:

ß-Thalassemia

Red cell aplasia (Diamond-Blackfan syndrome) unresponsive to pharmacologic therapy

Platelet

- Whole blood-derived platelet concentrates separated within 8 hours of collection, contain a minimum of 5.5×10^{10} platelets

- Platelets apheresis.

  contain a minimum of 3×10^{11} platelets

  suspended in 200-300 mL of plasma.

PLATELET TRANSFUSION IN PATIENTS < 4 MONTHS OF AGE

- Platelet count < 20-30,000/μL (20-30 x 109 /L) in infants lacking platelet production
- Platelet count < 50,000/μL (50 x 109 /L) with bleeding, or prior to a non-neurologic invasive procedure or minor surgery
- Platelet count < 100,000/μL (100 x 109 /L) in a sick premature infant or prior to a neurologic invasive procedure or surgery, cardiovascular surgery, or other major surgery
- Qualitative platelet defect with bleeding, prior to an invasive procedure or surgery, or with unexplained excessive bleeding during cardiopulmonary bypass
- Platelet count < 80,000-100,000/μL (80-100 x 109 /L) prior to or during an ECMO procedure, or with unexplained excessive bleeding during the procedure.

PATIENTS ≥ 4 MONTHS OF AGE

- Prophylaxis in a patient with a platelet count < 10,000/µL (10 x 109 /L) due to hypoproliferative thrombocytopenia;
- Prophylaxis in a patient with a platelet count < 50,000/µL (50 x 109 /L) prior to an invasive procedure if that procedure cannot be postponed.
- A platelet count ≥ 100,000/µL (100 x 109 /L) is recommended prior to neurosurgical and some ophthalmologic procedures
- Platelet count < 80,000-100,000/µL (80-100 x 109 /L) prior to or during an ECMO procedure, or with unexplained excessive bleeding during the procedure.
- Administration of platelets in massive transfusion
- Platelet dysfunction with bleeding or prior to an invasive procedure

- In the absence of life-threatening bleeding, platelet transfusion is not indicated in cases of immune thrombocytopenia purpura (ITP), post-transfusion purpura (PTP), thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome (HUS), or heparin-induced thrombocytopenia (HIT)

PLATELET DOSING AND ADMINISTRATION

- The usual dose of platelets is 5-10 mL/kg (the same dose for pooled platelet concentrates and apheresis platelets)
- The rate of infusion depends on the type of component, the total volume to be infused, venous access, and the patient's intravascular fluid tolerance.
- Total duration of administration for any component or pool must not exceed 4 hours.
- ABO and Rh compatible with the recipient's red blood cells, except in emergent situations.

ASSESSMENT OF RESPONSE TO PLATELET TRANSFUSION

- Transfusion of usual dose is expected to increase the platelet count by 50,000-100,000/μL (50-100 x 10^9) in a non-refractory patient, depending on the actual number of platelets present, which is variable based on the preparation method.
- In order to assess the patient's response to the transfusion, a post-transfusion platelet count should be performed within 60 minutes after completion of the transfusion

CORRECTED COUNT INCREMENT

CCI = (posttransfusion count - pretransfusion count) x body surface area (m^2) /of platelets transfused

Example:

2 kg infant receiving 10 mL/kg (20 mL)
Apheresis platelet unit = 3×10^{11}/300 mL x 20 mL = 2.0×10^{10} platelets transfused

WB-derived platelet unit = 5.5×10^{10}/50 mL x 20 mL = 2.2×10^{10} platelets transfused

REFRACTORINESS TO PLATELET TRANSFUSION

- The response to platelet transfusions should be monitored by obtaining a platelet count 10-60 minutes after each transfusion.
- Poor post-transfusion platelet count increments (CCI < 7.5×10^9 /μL) following 2 or more consecutive transfusions of ABO-compatible platelets indicate platelet refractoriness.

DISEASES REQUIRING INTERMITTENT OR CHRONIC RBC TRANSFUSION IN CHILDHOOD

- Chronic renal failure
- Thalassemia
- Sickle cell disease
- Aplastic anemia
- Oncologic(Leukemia, solid tumor)
- Diamond-Blackfan anemia
- Transient erythroblastopenia

TRANSFUSION RISKS

- Infectious
  - Viral
  - Bacterial
- Noninfectious
  - Reaction to RBC Antigens
    - Acute Hemolytic Transfusion Reactions (AHTR)
    - Delayed Hemolytic Transfusion Reactions (DHTR)
  - Reactions to Donor Proteins
    - Minor Allergic Reactions
    - Anaphylactic Reactions
  - White Cell-Related Transfusion Reactions
    - Febrile Reactions
    - Transfusion-Related Acute Lung Injury (TRALI)

TRANSFUSION REACTIONS

- Any unfavorable transfusion related
- event occurring in a patient during or
- after transfusion of blood components.
  - Acute: may occur within minutes or hours
  - of beginning the transfusion.
  - Delayed: when a transfusion Rxn
  - manifests itself a few days after
  - the transfusion

REACTIONS

- Immune mediated
- Non- immune mediated

TRANSFUSION COMPLICATIONS

- Fluid overload
- Reactions
- Transfusion transmitted diseases

TRANSFUSION-ASSOCIATED CIRCULATORY OVERLOAD (TACO)

- Risk factors
  - Patients with limited cardiopulmonary reserve (very young and elderly)
  - High volume transfusion
  - History of cardiac or renal disease
- Onset: within 1-2 hours after transfusion
- S/S: shortness of breath, cough, tachycardia, cyanosis, chest tightness, volume overload (JVD, S3 gallop, peripheral edema)
- Tx: supplemental O2, diuretics or other means of removing volume
- Prevention: slow administration of blood, pretreatment with diuretics (or blood administration with dialysis)

deltaco.com

IMMUNE REACTIONS

- Hemolytic (acute and delayed)
- Non-hemolytic (includes febrile, uriticarial, anaphylactic, purpura, etc)
  - These are primarily due to the sensitization of the recipient to donor blood cells (either red or white), platelets or plasma proteins.
  - Less commonly, the transfused cells or serum may mount an immune response against the recipient.

NON- HEMOLYTIC RXNS

- Due to sensitization of recipient to donor WBC, platetes or plasma proteins.
  - Febrile
  - Urticarial
  - Anaphylactic
  - Pulmonary edema (non-cardiogenic)
  - GVHD
  - Immune suppression

FEBRILE NON-HEMOLYTIC RXNS

- Due to cytokine released from leukocytes during storage or infusion
- Relatively common
- Rise in temperature by $\geq 1^{0c}$ during or within 24 hrs of the completion of the transfusion.
- Flu- like symptoms of chills, cold sensation, rigor (shaking) and in some cases headache and nausea are also present.
- In 3-7% of patients receiving RBC.
- More common in platelet transfusion (20-30%)

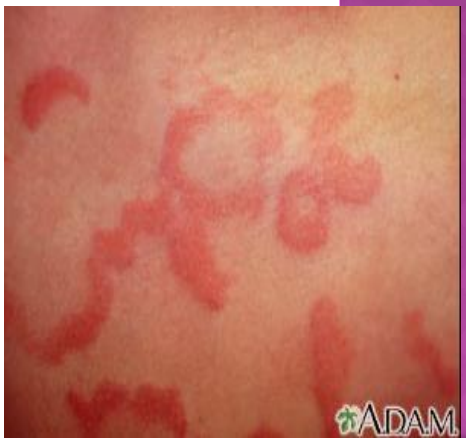
TREATMENT OF FNHTR

- Stop transfusion, antipyretic Acetaminophen)

- Severe → narcotic (for rigors), morphine 0.1 mg/kg IV, antibiotic or both

URITICARIAL (ALLERGIC RXN)

- Most common complication.
- Symptoms: hives and itching without fever, dizziness, headache and difficulties in breathing.
- In 1% of all transfusions.
- Due to sensitization against plasma proteins



TREATMENT

- Localized uriticaria:
  - Transfusion interruption, antihistamine.
  - Resolution within 30 min → transfusion
  - Diphenhydramine 6.25-25 mg IV in 10-20 min
- Recurrent → pre-transfusion antihistamine.
- Severe (bronchospasm, laryngospasm, hypotension, shock)
- Methylprednisolone: 1-2 mg/kg IV push

ANAPHYLACTIC RXN

- Rare (1/150000 transfusions)
- Severe, can occur with very small amounts of blood.
- Typically in IgA deficient patients with IgA antibodies (1 of 600-800 patients in the general population)
- Treatment :
  - a- epinephrine
  - b- fluids
  - c- corticosteroid
  - b- supportive measure
- Washed packed RBC or deglycerolized frozen RBC or IgA free blood units.

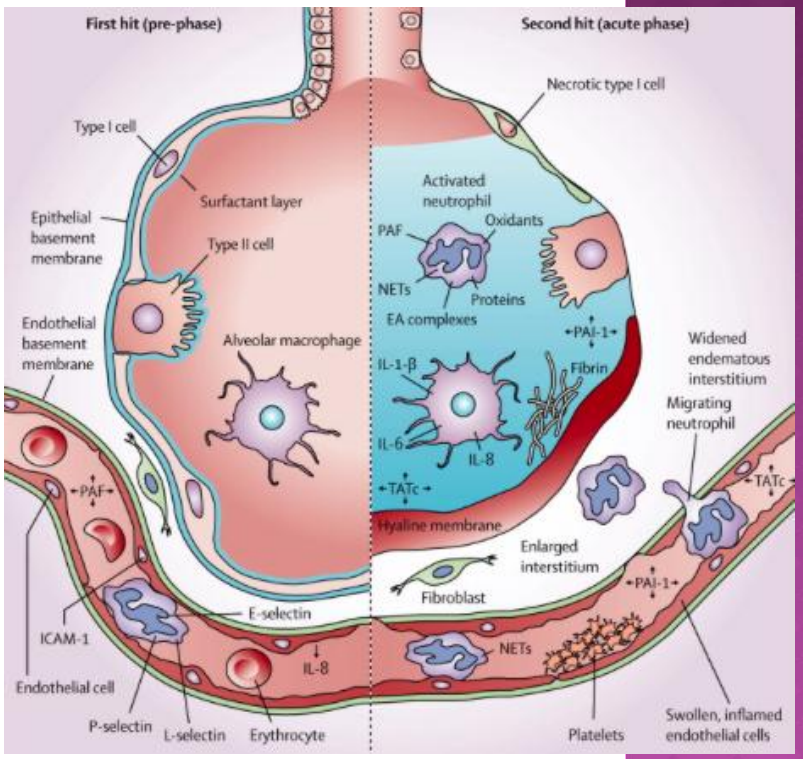
TRANSFUSION-RELATED ACUTE LUNG INJURY (TRALI)

- TRALI has an associated mortality of 5% and is the most common cause of transfusion-related death
- The figure demonstrates pathophysiology of TRALI, which is basically as follows:

- Recipient neutrophils are activated against an antigen in donor blood product →
- PMNs become sequestered in lung capillaries →
- Capillary leak →
- Pulmonary edema

TRANSFUSION-RELATED ACUTE LUNG INJURY (TRALI)

- Onset: during or within 6 hours of transfusion
- S/S: hypoxia, dyspnea, fevers, hypotension, pulmonary edema
- Treatment: stop the transfusion!
  - Supportive (may need intubation), O2
- Prevention: notify blood bank of reaction



thelancet.com

TRANSFUSION-RELATED ACUTE LUNG INJURY (TRALI)

- TRALI presents as sudden onset of respiratory difficulties during or shortly after transfusion. Hypoxemia and lung infiltrates are detected on chest X-rays in almost all patients with TRALI, and half of patients show a pinkish, frothy sputum [25]. Tachypnea, tachycardia, and elevated airway pressure are frequently observed. Fever, hypotension, and cyanosis occur in less than one-third of patients with TRALI.
- Confirming hypoxemia, obtaining a chest X-ray, and evaluating vital signs are required to diagnose TRALI.
- No laboratory test is specific for diagnosing TRALI.
- Blood bank should investigate associated donors for presence of anti-human leukocyte antigen (HLA) and possibly anti-human neutrophil antigen (HNA) antibodies. Goal is to find donors should be deferred from future donations.

TRALI VERSUS TACO

	TRALI	TACO
Blood pressure	Low-normal	Normal-high
Body temperature	Normal-elevated	Normal
CXR	No vascular congestion	Vascular congestion, pleural effusion
BNP	Low (< 250 pg/ml)	High
PAOP	Low-normal	High
Ejection fraction	Normal function	Abnormal function
Response to diuretics	Inconsistent	Improved
Edema fluid	Transudate	Exudate

Kim et al. 2015.

DIAGNOSTIC CRITERIA

- Acute onset of hypoxemia (within 6 hours of conclusion of transfusion)
- Bilateral CXR infiltrates consistent with ALI
- Absence of evidence of left atrial hypertension
- Absence of temporally related causes of ALI

TREATMENT

- Largely supportive
- Transfusion should be stopped if recognized in time
- Supplemental oxygen and ventilation support provided if necessary
  - Use low tidal volume settings like in ARDS
- No diuretics
- Glucocorticoids have been administered but no evidence supporting their administration

GVHD

- Life threatening
- donated WBC destroy the recipients cells
- Exclusively in immunocompromised patients, who received cellular blood products containing lymphocyte
- Immune response of lymphocyte
- Pre- transfusion irradiation inactivates lymphocyte.

POST - TRANSFUSION PURPURA

- Common
- Platelet antibodies development
- May lead to profound thrombocytopenia which usually occurs about one wk post transfusion
- Recommended treatment:
  Plasmapheresis

IMMUNE SUPPRESSION

- leukocyte- containing blood products appears to be immunosuppressive, causing a decrease in Nk cell function , decreased phagocytosis and decrease T-suppressor/T-helper. (Immunomodulation)
- Unclear reason
- Leukocyte reduction is beneficial
- Many lead to life –threatening events, such as multiple organ failure, ↑risk of infection after surgery and recurrence of certain malignancies.

HEMOLYTIC REACTIONS

- Usually involve the destruction of transfused blood cells by the recipients antibodies can cause hemolysis of the recipient blood cells.
- Two types :
  1- Acute (intravascular)
  2- Delayed (extra vascular)

ACUTE HEMOLYTIC (ATHR) RXNS

- Usually due to ABO mismatch
- Most commonly in operating room
- 1 in 25000 transfusions
- Often very severe
- Occounts for > 50% deaths related to transfusion
- Diagnosis: positive coomb's

ACUTE HEMOLYTIC TRANSFUSION REACTIONS

- Over 300 antigens on human RBC's
- Most common antibodies that fix complement
  - A, B, Kell, Kidd, Duffy
- Rh antibodies do not fix complement but can cause serious hemolysis

AHTR PATHOPHYSIOLOGY

- Antibodies and complement in recipient plasma attack antigens on donor RBC's causing hemolysis
- Antigen-antibody complexes activate Hageman factor (factor XII) producing bradykinin leading to capillary permeability and hypotension
- Complement system releases histamine and serotonin from mast cells resulting in bronchospasm
- 30-50% of patients will develop DIC

AHTR PATHOPHYSIOLOGY

- Hemolysis releases hemoglobin (Hb)
- Hb binds to haptoglobin and albumin initially
- Will circulate unbound until excreted by kidneys
- Renal damage causes
  - Systemic hypotension and renal vasoconstriction
  - Free Hb form acid hematin damaging renal tubules
  - Antigen-antibody complexes may deposit in glomeruli

SIGNS AND SYMPTOMS

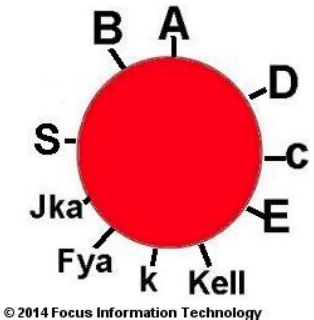
- Fever
- Chills
- Nausea and vomiting
- Diarrhea
- Rigors
- Hypotension and tachycardia (bradykinin)
- Flushed and dyspneic (histamine)
- Chest and back pain (cytokine release)
- Headache
- Feeling of impending doom
- Hemoglobinuria eventually oliguria

SYMPTOMS

- Awake patients: Fever, Chills, nausea, chest and flank pain
- Anesthetized: ↑Temp, Unexplained tachycardia, hypotension, hemoglobinuria, DIC, oozing in the surgical field, shock and renal shutdown.
- May be minor symptoms

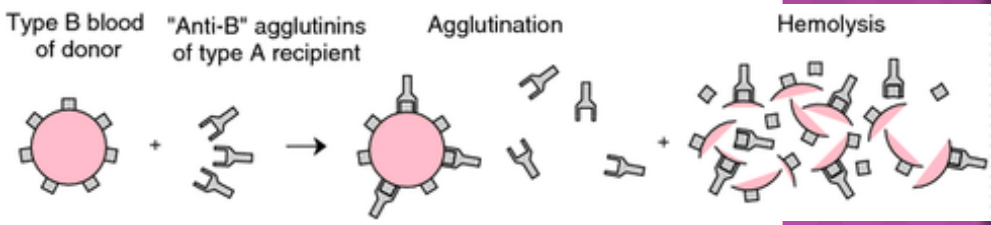
DELAYED HEMOLYTIC TRANSFUSION REACTION

- Onset of symptoms: 5-10 days after RBC transfusion
- S/S: hemolytic anemia, jaundice, fever (can also be asymptomatic)
- Life-threatening complications are rare
- Confirmation: repeat type and screen to detect alloantibody
- Treatment: supportive



Acute Hemolytic Transfusion Reaction

- Abrupt onset of S/S
- S/S: intravascular hemolysis, hypotension, fevers, AKI, pain at the infusion site, DIC, pink plasma or urine
- Treatment: stop the transfusion!
  - Send blood back to blood bank to check for incompatibility, hemolysis
  - Supportive treatment with IVF, vasopressors, diuresis



http://arimmuneresponseassignment.weebly.com/report.html

DIAGNOSIS

- Stop transfusion
- Recheck patient and unit labeling
- Examine centrifuged plasma sample for pinkish discoloration representing free Hb
- Hemolysis should be assumed to be hemolytic transfusion reaction until proven otherwise
- Notify blood bank
- Aseptically seal unit and return
- Coombs test
  - Examines recipient RBC's for presence of surface immunoglobulins and complement

TREATMENT

- Maintain systemic blood pressure
  - Deliver volume
  - Vasopressors
  - Inotropes
- Preserve Renal function and urine output
  - Administering fluids fluid (20cc/kg normal saline)
  - Diuretics (mannitol or furosemide)
  - Sodium bicarbonate to alkalinize urine
- Prevent DIC
  - No specific therapy
  - Prevent hypotension and support cardiac output
  - Decreases stasis

DELAYED HEMOLYTIC RXNS

- Generally mild
- Due to non-D antigens of Rh system or minor blood groups such as kell, Duffy or kidd Ags.
- 1-1.6% chance of developing Abs.
- One in 2500 transfusion, most often in females with previous exposure secondary to pregnancy.

DELAYED HEMOLYTIC TRANSFUSION REACTIONS

- Compatible RBC's are rapidly eliminated within days
- Typically due to donor RBC antigen to which recipient has been previously exposed via transfusion or pregnancy
- Over time antibody levels fall too low to be detected
- With re-exposure anamnestic response results in antibodies and lysis of foreign RBC's
- Coated RBC's are sequestered extravascularly (spleen and reticuloendothelial system) and lysed

- Two types:

  1- Primary

  2- Anamnestic

- Delayed hemolytic transfusion reactions:

  a. Primary immunization (mild, unexpected ↓in Hb 2-3 wk later)

  b. Anamnestic response (sensitization to one or more Ag in prior transfusion or pregnancy)

  3-10 days after transfusion, profound anemia, bilirubinemia milder than ABO incompatibility.

  diagnosis: positive coombs.

- Symptoms: maliase, jaundice, fever, a fall in HCT, ↑unconjugated bilirubin

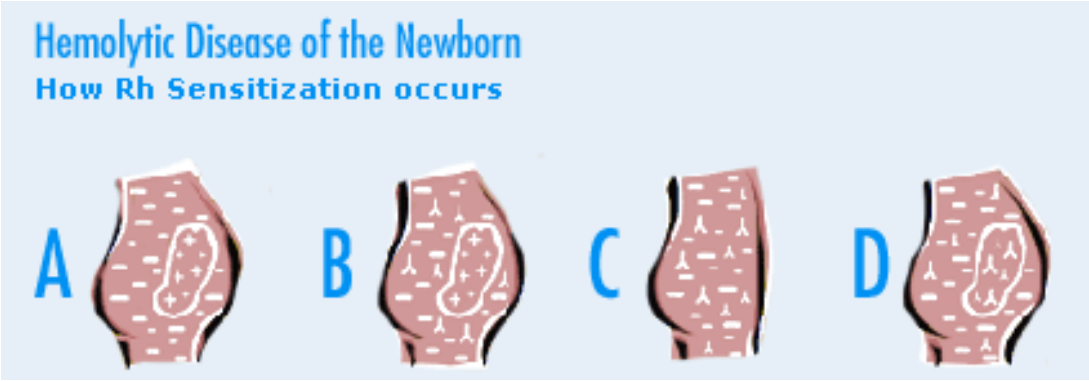
- Diagnosis: +ve coombs test
- Treatment : supportive

DIAGNOSIS AND TREATMENT

- Usually detected in the first or second week
- Low-grade fever
- Increased indirect bilirubin
- Unexplained reduction in Hb
- Decreased serum haptoglobin
- Confirmed by positive Coomb's test
- Resolves as transfused cells are removed
- Monitor Hb
- Maintain hydration
- Re-transfuse if necessary

HEMOLYTIC DISEASE OF THE NEBORN - HOW IT OCCURS

- A child is Rh pos
- B during pregnancy fetal Rh pos rbc's escape into maternal circulation
- C Mother produces antibodies to Rh (D) antigen
- D Second pregnancy with Rh (D) pos child results in destruction of fetal D pos rbcs



SUMMARY

- It is important to recognize the possible reactions that can be associated with blood transfusions
- If you suspect a reaction, stop the transfusion and assess the patient's vital signs, signs and symptoms as some reactions may be life-threatening
- Notify the blood bank if serious reactions are suspected

TAKE HOME MESSAGE

Using appropriate transfusion strategy of transfusion and monitoring Of patients are very important in improving health related quality of Life.

Thank you very much for your attention!!!