Transfusion reaction	Pathology	Presentation	Prevention/treatment
Acute hemolytic	RBC antibodies (vs ABO) that activate complement	Intravascular hemolysis w/ hemoglobinemia, hemoglobinuria, Fever, pain, nausea, hypotension, dyspnea, renal failure, DIC, death Onset w/in 24 hrs	Proper labeling
Delayed hemolytic	IgG RBC antibodies (vs Rh) that do not activate complement	Extravascular hemolysis w/ falling hematocrit and hyperbilirubinemia Fever, leukocytosis, asymptomatic Onset w/in 1 day – wks	Usually not detectable by routine pretransfusion testing
Febrile non-hemolytic	Common in multiply transfused patients due to donor cytokines accumulated during storage or recipient antibodies to donor leukocytes	Fever	
Allergic	Common in multiply transfused patients and may be due to recipient antibodies to donor plasma proteins	Urticaria, flushing, itching, nausea, vomiting, dyspnea	
Anaphylaxis	Common in Ig-A deficient patients w/ anti-Ig-A as a result of prior transfusion	Anaphylaxis	RBC & platelets washed free of plasma proteins
Bacterial contamination	Yersinia & pseudomonas growth in refridgerated cells, gram +/- growth in platelet concentrates at room temp	Septic shock High mortality rate	Unpredictable, little can be done
Transfusion-related acute lung injury (TRALI)	Neutrophil activating substances accumulated during storage or donor anti-leukocyte antibodies vs recipient WBC cause leakage of capillaries	Non-cardiogenic pulmonary edema during or after transfusion 10% mortality rate Resolves w/in 48-72 hrs, does not progress to ARDS	Pre-storage leukocyte depletion of blood components
Post-transfusion purpura	Primary immune response to a platelet specific ag (initial cross-reaction w/ auto and allo platelets, but eventual narrowing of specificty to allo only)	Profound thrombocytopenia 1-3 wks post-transfusion, returning to normal 2-3 wks after onset	Restriction of activities, IVIg if bleeding occurs (high risk of intracranial hemorrhage)
Non-immune hemolysis	Cold, heat, hypotonic solutions, small-bore needles resulting in transfusion of hemolytic blood	Hemoglobinemia, hemoglobinuria, hyperkalemia & renal failure	Recognition to prevent additional transfusion of hemolyzed blood
Hypotensive	Bradykinin (potent vasodilator) generation during transfusion	Hypotension, tachycardia Patients on ACE inhibitors at risk	Stop transfusion and resume at slow rate when blood pressure stabilizes
Transfusion-associated GVHD	Donor T-cells engraft and recognize recipient as foreign, even in transfusion from 1st-degree relative	Pancytopenia, fever, rash, diarrhea, liver dysfunction wks after transfusion, esp patients w/ CMI def Death from infection or bleeding	Gamma irradiation of blood components to prevent lymphocyte proliferation

Neonatal disorder	Pathogenesis	Presentation	Treatment/prevention
Hemolytic disease of the newborn (HDN)	Maternal IgG crosses placenta and binds fetal RBC, which are cleared by spleen (RhD, severe) or complement (ABO, unnoticeable)	Hemolytic anemia, hydrops fetalis, hyperbilirubinemia (can cross BBB and cause kernicterus, a lethal encephalopathy), hypoglycemia (pancreatic island hyperplasia), hepatosplenomegaly (compensatory erythropoiesis) Previous pregnancy required	Treatment: early delivery, intrauterine transfusion, exchange transfusion (hb<14, bilirubin>4), phototherapy (reduces bilirubin), maternal plasmaphoresis Prevention: antibody screen 1st prenatal visit & 28 wks, Rh Ig (rhogam) at 28 wks, delivery, and at intervention
Neonatal alloimmune thrombocytopenia (NAIT)	Maternal antibody vs fetal platelets (HPA-1, rarely HLA)	Similar to HDN Thrombocytopenia at birth (but resolves in 7 days as maternal antibodies cleared), risk of intracranial hemorrhage May occur during 1st pregnancy	Treatment: ag negative platelet transfusion (maternal donor) Prenatal management: no prophylaxis, IVIg, fetal genotyping for pre-delivery maternal platelet donation