RBC Hemolysis

1. Anemia: *normocytic*, normal WBCs, *increased reticulocytes* (polychromasia)
2. Jaundice/scleral icterus (due to high unconjugated bilirubin in blood)
3. Splenomegaly (excessive recycling/processing of RBCs by splenic macrophages)

Labs:
→High LDH & indirect bili
→low free serum haptoglobin
→hemoglobinuria (red pee)
→Hemoglobinemia (red plasma)

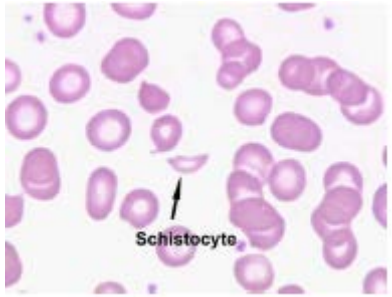
Extracorpuscular Causes (usually acquired)

Immune Hemolytic Anemia
→Caused by Ig's and Complement
→(positive DAT)

Auto-immune
→Warm Abx (IgGs; *no* fam Hx of hemolysis, idiopathic or 2^0 other autoimmune dx)
→Cold Abx (IgM, C3; active @ 2-4^0C, acrocyanosis; often due to viral infection)
Tx: immunosuppression, tx underlying cause

Allo-immune
→RBC antigen mismatch (hemolytic transfusion rxn)
→Drugs

Non-Immune Hemolytic Anemia
→MAHA (TTP, DIC, HUS, artificial valves; blood film: schistocytes and no platelets)
→Malaria
→Hypersplenism



Intracorpuscular Causes (usually hereditary)

RBC membrane defects

Hereditary spherocytosis
→Autosomal dominant
→1/5000
→Spectrin/ankryin deficiency -> shedding of RBC membrane ->*spherocytes*
→+ family Hx of "anemia + jaundice"
→Dx: Osmotic Fragility Test
→**Tx:** splenectomy if anemia is severe

RBC metabolic defects

G6PD deficiency
→Common (400 million worldwide)
→X-linked
→Deficiency = protects against malaria
→**Heinz bodies** (denatured HgB)
→**Bite Cells** (where Heinz-bodies were removed by spleen)
→Dx: G6PD assays (may be inapprop. Normal due to reticulocytosis)
→**Tx** : eliminate infections, oxidative foods, drugs → anemia = self-limiting

Pyruvate Kinase deficiency

Defective RBC contents (Hgb)

Thalassemia

Hemoglobinopathies