Hemolytic Anemia

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Learning Objectives: As a result of participating in this activity, the learner will be able to:

• Identify the general diagnostic findings of hemolytic anemia
• Recognize the types of hemolytic anemia
• Discuss the appropriate treatment of hemolytic anemia
### Anemia

**Normal CBC parameters in adults**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>13.6 to 16.9</td>
<td>11.9 to 14.8</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>40 to 50</td>
<td>35 to 43</td>
</tr>
<tr>
<td>RBC</td>
<td>4.2 to 5.7</td>
<td>3.8 to 5.0</td>
</tr>
<tr>
<td>MCV</td>
<td>82.5 to 98</td>
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<tr>
<td>Retic</td>
<td>0.5-2%</td>
<td></td>
</tr>
<tr>
<td>PLT</td>
<td>152-361</td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>3.8 to 10.4</td>
<td></td>
</tr>
</tbody>
</table>
## Quick Evaluation in Anemia

Always review the history and perform physical exam  
Will need CBC and look at MCV and retic at first

<table>
<thead>
<tr>
<th>MCV &lt;80</th>
<th>MCV 80-100</th>
<th>MCV &gt;100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Check for iron deficiency</td>
<td>ACD, Infection, Medication, hypothyroid</td>
<td>Vitamin B12/folate medication, ETOH</td>
</tr>
<tr>
<td>Thalassemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>If ferritin &lt;50 replace with IV iron</td>
<td>If ferritin &gt;50 Hgb electrophoresis</td>
<td></td>
</tr>
</tbody>
</table>
# Medication and mechanisms associated with Anemia

<table>
<thead>
<tr>
<th>Types of anemia</th>
<th>Mechanism</th>
<th>Examples of medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron deficiency</td>
<td>GI irritation</td>
<td>ASA, NSAIDS, Steroids</td>
</tr>
<tr>
<td>VB12 deficiency</td>
<td>Impaired absorption</td>
<td>PPI, H2 blocker, antacids</td>
</tr>
<tr>
<td>Folate deficiency</td>
<td>Interference with metabolism</td>
<td>MTX, certain antibiotics, antiseizure med</td>
</tr>
<tr>
<td>Impaired BM</td>
<td>Impaired DNA synthesis</td>
<td>Hydroxyurea and chemo</td>
</tr>
<tr>
<td>Hemolysis</td>
<td>Immune medicated</td>
<td>Antibiotics</td>
</tr>
<tr>
<td></td>
<td>Oxidative stress</td>
<td>Antibiotics</td>
</tr>
<tr>
<td></td>
<td>Methemoglobinemia</td>
<td>Benzocaine, lido nitrates</td>
</tr>
<tr>
<td></td>
<td>Thrombotic microangiopathy</td>
<td>Chemo, immune sup</td>
</tr>
</tbody>
</table>
Evaluate for other medical conditions that cause anemia

- Cancer
- Chronic conditions such as chronic kidney disease which can cause low erythropoietin and inflammation.
- Rheumatologic conditions
- Hypothyroid
- Infections
Hemolytic Anemia

• Hemolysis is the destruction of red blood cells (RBC's), leading to decreased RBC survival.
• The bone marrow's response to hemolysis: increased erythropoiesis, reflected by reticulocytosis.
• If the bone marrow is unable to completely compensate for hemolysis, then anemia occurs.
Why do RBC lyse? How are RBC removed?

- Normal aging
- Antibody binding
- Complement activation
- Phagocytic clearance

Extravascular

Intravascular
Activated Macrophages

- Large phagocytic WBC
- Essential to immune response
- Bind to RBC surface proteins
- Receive “eat” me signals
- Engulfed RBC’s are removed from the circulation
- Processed in the spleen and the liver
Identify the general diagnostic findings of hemolytic anemia

• Any recent blood transfusions?
• Peripheral smear, CBC, Retic, LDH, Haptoglobin and indirect bilirubin.
  o Peripheral smear: looking for spherocytes, schistocytes, bite cells, sickle cells,
  o CBC: Hgb level, any other cytopenias?
  o CMP: Cr, bilirubin
  o Retic: is it elevated?
  o LDH: is it elevated?
  o Haptoglobin: is it undetectable?
  o Indirect bilirubin: is it elevated?
• Urinalysis: heme or hemosiderin? Dark/red in color?
• If above all “yes” next step is ordering Coomb's test also known as direct antiglobulin test DAT
Clinical Symptoms

- Pallor
- Fatigue
- Jaundice
- Dark urine
- Splenomegaly

Common clinical findings and laboratory markers

- Pallor
- Fatigue
- Jaundice
- Dark urine
- Others signs (infections, organomegalies, skin rashes, arthralgia, etc.)

Warm AIHA
- DAT+ for IgG/IgG+C
  - Younger age and female predominance
  - Acute presentation
  - Severe anemia
  - Almost all patients require treatment
  - Deep asthenia, palor, jaundice
  - Angina, systolic murmur
  - LDH +/+/
  - More rapid response to steroids

Cold AIHA
- DAT+ for C3d
  - Older age
  - Chronic presentation
  - LDH+++/
  - Milder anemia
  - +20-30% Hb >10g/dL
  - and never requires treatment
  - Hemoglobinuria
  - Peripheral circulatory symptoms (acrocyanosis, Raynaud, rash)
  - Bone marrow lymphoid infiltrate may be present
Direct and Indirect Coombs test

- Helps to differentiate immune vs non-immune mediated hemolysis
- Coombs test or Direct antibody test
  - Looking for antibodies attached to the red blood cell
    - The patient’s blood is collected in a EDTA tube to prevent complement factor C3 form attaching to the red blood cell. Laboratory washes the RBC then adds a reagent to detect bound IgG and/or Complement C3 (also evaluates IgM)
  - Testing will also be completed in warm temperature as well as cold
- Positive test alone does NOT = hemolysis
- With indirect Coombs test this occurs before a pt receives a blood transfusion
Recognize the types of hemolytic anemia

Hemolytic Anemia Types

• Acquired
  o Immune
    • AIHA
    • Cold agglutinin
    • Paroxysmal Cold Hemoglobinuria
  o Non-immune
    • Clonal disorders: Paroxysmal Nocturnal Hemoglobinuria
    • Secondary due to renal and liver disease
    • Medication induced
    • Transfusion related
    • Microangiopathy
Recognize the types of hemolytic anemia

Hemolytic Anemia Types

• Hereditary
  o Hemoglobin defects in sickle cell and thalassemia
  o RBC membrane and metabolic defects
Acquired Hemolytic Anemia

- Autoimmune hemolytic anemia
  - Warm AIHA
  - Cold agglutinin disease (CAD)
  - Paroxysmal cold hemoglobinuria
- Paroxysmal nocturnal hemoglobinuria
- Drug induced
- Transfusion related
Warm AIHA

- Destruction of RBCs due to presence of warm agglutinins (almost always IgG) which bind IgGs over RBC surface at body temperature
- IgG-coated RBCs are recognized by macrophages: phagocytosis of entire RBC or part of the membrane -> spherocytes -> Spleen trapped/destruction
- IgG also leads to antibody-dependent cell-mediated cytotoxicity and activation of complement
Warm-autoantibody type: autoantibody maximally active at 37 degrees Celsius

- Primary or Idiopathic
- Secondary
  - Immunodeficiency disorders: CVID
  - Infections: HIV, CMV, HSV, Hepatitis E, babesiosis (if prior splenectomy)
  - Autoimmune/connective tissue disorders: SLE, RA, autoimmune lymphoproliferative syndrome (ALPS)
  - Lymphoproliferative disorders: HL, NHL, CLL
  - Medications: Penicillin, cephalosporin, ICIs, methyldopa
  - Pregnancy
  - Babesiosis associated with AIHA in asplenic patients
  - Sickle cell disease = bystander hemolysis
  - Solid organ transplant recipients who receive an ABO-compatible, but not identical, allograft can develop hemolysis due to passenger lymphocyte syndrome (donor lymphocytes present in the transplanted organ that react with recipient RBCs)
Warm AIHA

- Patient may have a sudden onset of anemia/jaundice

Work-up:
- CBC: Anemia, may have elevated MCV
- Reticulocyte count: elevated
- PBS: spherocytes
- Hemolysis panel: elevated LDH, low haptoglobin, elevated indirect bilirubin
- Direct coombs test: **positive for IgG and or C3** (vs. cold agglutinin only positive C3)
Peripheral blood smear

Peripheral smear in severe autoimmune hemolytic anemia

Peripheral blood smear from a patient with Coombs-positive autoimmune hemolytic anemia. The smear shows the presence of many spherocytes (arrowheads), one nucleated red blood cell (dashed arrow), and a number of larger polychromatophilic red cells (arrows), representing a reticulocytosis in response to the anemia.

Courtesy of Carola von Kapff, SH (ASCP).

Normal peripheral blood smear

High-power view of a normal peripheral blood smear. Several platelets (arrowheads) and a normal lymphocyte (arrow) can also be seen. The red cells are of relatively uniform size and shape. The diameter of the normal red cell should approximate that of the nucleus of the small lymphocyte; central pallor (dashed arrow) should equal one-third of its diameter.

Courtesy of Carola von Kapff, SH (ASCP).
Management of Warm AIHA

- Blood Tx: severe anemia (<6), HD unstable, AMS, chest pain
- Transfusion type-specific blood, even if not crossmatch-compatible (autoantibodies will react with all donor RBCs)
  - Transfuse “best matched” blood; ABO identical or compatible blood products
  - DO NOT withhold blood for concern of incompatibility
- Replete any deficiencies i.e. folic acid.
- Treat underlying etiology if present (medication, infection...)
- 1st line Treatment:
  - Prednisone 1-2mg/kg/day PO or methylprednisolone equivalent IV
  - Dose given until Hb >10 (usually within 2-3 weeks)
  - Taper over 4-6 months to prevent early relapses
- 2nd line Treatment:
  - No response to initial treatment, hemoglobin <10 g/dL despite therapy, early relapse, and/or inability to taper prednisone to ≤10 mg daily to maintain a higher hemoglobin level.
  - Rituximab can be added to Glucocorticoid
Cold Agglutinin Disease

- Not a common disease (1 per 1,000,000)
- Older female, age > 70 y, cold-induced symptoms (90%): Raynaud’s, acrocyanosis, livedo reticularis. Cutaneous ulceration, Anemia Symptoms, Extravascular hemolysis
- Thermal amplitude is in the range of 37°C (ie, refrigerator temperature), but many antibodies are active at higher temperatures such as occur in colder areas
- Cold agglutinins are antibodies (IgM) that recognize (RBC) antigens at temperatures below normal body temperature.
- Causes
  - Primary CAD: originate from a low-grade lymphoproliferative disorder and thus to be monoclonal.
  - Secondary:
    - Infection: *M. pneumoniae* (atypical pneumonia) or EBV
    - Lymphoid malignancy (monoclonal IgM gammopathy)
    - Autoimmune disorders
Cold Agglutinin Disease

- Positive direct antiglobulin (Coombs) test (DAT)
- Direct Coombs positive for IgM complement C3 and negative for IgG.
- Causes hemolysis by fixing Complement
- Cold agglutinin titer of >1:64 at less than 37 degrees (high titer required to be clinically significant)
- Peripheral blood smear in CAD shows RBC agglutination and evidence of hemolysis
Treatment of Cold Agglutinins Disease

1. Will need to avoid being cold
2. Treat underlying disease when indicated (e.g. antibiotics, antivirals)
3. If anemia is severe transfusions should be given through a blood warmer (cannot be above 40°C) RBCs +/- plasmapheresis (temporary measure) or IVIG (critical hemolysis)
4. If pharmacological treatment is needed this is usually caused by a lymphoproliferative disorder.
   - Rituximab monotherapy is first choice.
   - Glucocorticoids and splenectomy are not effective therapy in the majority of patients with CAD
# Warm vs. Cold Hemolytic Anemia

<table>
<thead>
<tr>
<th>Warm Agglutinin</th>
<th>Cold Agglutinins</th>
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</thead>
<tbody>
<tr>
<td><strong>1) Idiopathic</strong></td>
<td><strong>1) Mycoplasma</strong></td>
</tr>
<tr>
<td><strong>2) Lymphoma, CLL</strong></td>
<td><strong>2) EBV (mono)</strong></td>
</tr>
<tr>
<td><strong>3) Drugs (eg: Methyl Dopa, Pencillin, Quinidine)</strong></td>
<td><strong>3) Idiopathic</strong></td>
</tr>
<tr>
<td><strong>4) Post-viral, HIV</strong></td>
<td><strong>4) Lymphoma</strong></td>
</tr>
<tr>
<td><strong>5) Immune deficiency (CVID)</strong></td>
<td><strong>CAUSES</strong></td>
</tr>
<tr>
<td><strong>6) CTD (eg: SLE)</strong></td>
<td><strong>Cold Agglutinins</strong></td>
</tr>
</tbody>
</table>

### Causes

- + Coombs (Direct Antiglobulin test) against IgG

### Diagnosis

- Spherocytes
- Peripheral smear findings: Agglutination

### Treatment

- 1) Avoid cold exposure
- 2) Rituximab
- 3) Interferon

- Splenectomy and steroids NOT used
Paroxysmal Cold Hemoglobinuria

- PCN autoantibodies (IgG) bind RBC surface (P antigen) and fix complement in cold temperatures -> intravascular hemolysis upon rewarming (due to activation of complement) the autoantibody dissociates
- Antibody polyclonal
- Causes:
  - Children (more commonly affected): following a viral infection, congenital syphilis
  - Adults: 3ry syphilis (more prevalent before 20th century), viral infections (VZV), autoimmune diseases, and rarely in lymphomas, CLL, post bacterial infection, post measles vaccine
- Clinical manifestations:
  - Intravascular hemolysis after cold exposure: dark urine, abdominal cramping, weakness/malaise, fever/chills, jaundice/pallor
  - Hemolysis is usually transient
Assessment and Testing

• Diagnosis:
  o Hemolysis lab changes
  o Intravascular features: free Hb in serum and/or urine
  o **Direct coombs only positive for C3 and not for IgG** (these only bind on cold temperatures)

• **Donath-Landsteiner test:**
  o Patient’s serum is cooled to 4°C (along with test RBCs) to allow for fixing of the autoantibody (if present) and complement. The sample is then warmed to 37°C to allow activation of complement and hemolysis.
  o Although is diagnostic, it is relatively insensitive and may rapidly become negative: Therefore a negative result does not exclude the disease. Test may be negative between episodes
Treatment

- Cold avoidance
- Transfusion (if severe anemia)- compatible unit challenging
- Hydration (to prevent AKI from intravascular hemolysis)
- Steroids are often used but data is limited
- Other options: Rituximab, Cyclophosphamide, eculizumab
- Splenectomy not indicated
- Rx (eg, syphilis, autoimmune or lymphoproliferative disorder)
- Autoantibodies have a half-life of two to three weeks; thus, even if therapy immediately halts production of the autoantibody, hemolysis may continue for two to three weeks.
## Medications with AIHA +/- +DAT

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>NSAIDS/analgesics</th>
<th>Chemotherapy</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>P-aminosalicylic acid</td>
<td>Atezolizumab</td>
<td>Nomifensine</td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>Penicillin</td>
<td>Bendamustine</td>
<td>Probencid</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Piperacil</td>
<td>Carboplatin</td>
<td>Puerarin (Chinese herb)</td>
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<tr>
<td>Cefazolin</td>
<td>Pyrimethamine</td>
<td>Cisplatin</td>
<td>Quinidine</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>Quinidine</td>
<td>Cisplatin</td>
<td>Quinine</td>
</tr>
<tr>
<td>Cefotetan</td>
<td>Quine</td>
<td>Cisplatin</td>
<td>Radiopaque contrast</td>
</tr>
<tr>
<td>Cefoxitin</td>
<td>Rifampin</td>
<td>Methotrexate</td>
<td>medium</td>
</tr>
<tr>
<td>Ceftazidine</td>
<td>Stibophen</td>
<td>Nivolumab</td>
<td>Tolbutamide</td>
</tr>
<tr>
<td>Ceftizoxime</td>
<td>Streptomycin</td>
<td>Oxaliplatin</td>
<td>Tramaterene</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>Teicoplanin</td>
<td>Pembrolizumab</td>
<td>Trimellitic</td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>Temafloxacine</td>
<td>Pemetrexed</td>
<td>anhydride (used in dyes, resins)</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>Tetracycline</td>
<td>Pentostatin</td>
<td></td>
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<tr>
<td>Cephalothin</td>
<td>Ticarcillin</td>
<td></td>
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</tr>
<tr>
<td>Chloramphenicol</td>
<td>Trimethoprim/</td>
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<tr>
<td>Ciprofloxacin</td>
<td>sulfamethoxazole</td>
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<td></td>
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<tr>
<td>Erythromycin</td>
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<tr>
<td>Isoniazid</td>
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<tr>
<td>Levofloxicin</td>
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<tr>
<td>Mefloquine</td>
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<tr>
<td>Nafcillin</td>
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Drug Induced AIHA

• There are many medications such as antibiotics, NSAIDS/analgesics, chemotherapy and others
• SELF LIMITED! If the medication is discontinued will usually resolve in 1-2 weeks
• MOST important is to discontinue the offending medication
• This occurs due to the medication injuring the RBC by antibody to medication, antibody to the RBC membrane or to both.
• If anemia is severe glucocorticoids and/or IVIG can be given.
Hereditary Hemolytic anemia

- Sickle Cell Anemia caused by hemoglobin structural variants

- Thalassemia which are disorders of decreased globin chain production

- Glucose-6-phosphate dehydrogenase (G6PD) caused by a genetic defect in the red blood cell (RBC) enzyme G6PD, which generates NADPH and protects RBCs from oxidative injury. G6PD deficiency is the most common enzymatic disorder of RBCs.
Sickle cell disease

- Sickle cell disease
  - Hemoglobin electrophoresis which will determine the percentage of Hgb A, Hgb S, Hgb C, Hgb A2, and Hgb F
  - CBC, Retic, LDH, and bilirubin, and ferritin
  - Review smear to see sickle cells
Thalassemia

• Thalassemia
  o Suspect if low MCV and no evidence of iron deficiency
  o CBC, review blood smear and iron studies
  o Hemoglobin electrophoresis
  o Hemoglobin gene sequencing for alpha thalassemia
Treatment

• Sickle cell crisis
  o Fluids
  o Pain medication
  o Oxygen
  o Transfusions if symptomatic
  o In vaso-occlusive crisis exchange transfusion is needed.
  o Recommend folic acid 1mg daily
  o No oral or IV iron
Treatment

- Thalassemia
  - If transfusion dependent will need to have RBC transfusions as needed
  - Should be receiving folic acid
  - No oral or IV iron
Hyper hemolysis Syndrome

- Development of severe Anemia
- Post-transfusion Hgb decrease the pre-transfusion Hgb
- May be subdivided into acute and delayed forms
  - Acute occurs within 7 days of transfusion
  - Delayed occurs later than 7 days, alloantibody formation
Who is at risk for Hyperhemolysis

- Underlying hemoglobinopathies
  - Sickle cell Disease
  - Thalassemias
- Other comorbidities requiring frequent transfusion
  - Myelofibrosis
  - Anemia of chronic disease
  - Lymphoma
Clinical presentation of hyperhemolysis

- Usually includes fever, jaundice and pain
- Elevated bilirubin
- Elevated LDH
- Decrease in reticulocytes
- Direct antiglobulin test (DAT) negative
- New alloantibodies may be present
- Recent history of transfusion
- Drops in Hgb and Hct despite transfusions
Bystander Hemolysis

- When native and donor cells are destroyed
- Complement deposition on the RBC
- Peripheral consumption and destruction via activated macrophages
- Major cause of anemia in hyperhemolysis
Management of Bystander Hemolysis

• Try to avoid future transfusions
• Supportive care
  o Monitor CBC
  o If transfusion is needed will need to try to find the best match
  o Monitor for renal failure.
Glucose-6-phosphate dehydrogenase (G6PD)

- Can have medication induced hemolysis (rasburicase etc).
- Hemolysis is seen in 2-4 days
- Stop medication and give supportive care.
Medication to avoid in G6PD deficiency

Chlorpropamide
Dabrafenib
Dapsone (diaminodiphenyl sulfone)
Fluoroquinolones (ciprofloxacin, moxifloxacin, norfloxacin, ofloxacin)
Methylene blue (methylthioninium chloride)$\Delta$
Nalidixic acid
Nitrofurantoin, nifuratel, and nitrofurazone (nitrofural)
Phenazopyridine (pyridium)
Primaquine and tafenoquine
Rasburicase and pegloticase
Sulfonylureas (eg, glipizide, glyburide [glibenclamide])
Food and Chemicals to avoid

- Fava beans
- Henna compounds (black and red Egyptian)
- Naphthalene (mothballs, lavatory deodorant)
- Phenylhydrazine
- "RUSH" (isobutyl nitrite, amyl nitrite)
Paryoxysmal Nocturnal Hemoglobinuria (PNH)

• PNH is an acquired clonal hematopoietic stem cell disorder caused by a somatic mutation of the PIGA gene that results in hematopoietic cells lacking GPI-linked proteins.
• Hemolysis in PNH is due to the action of complement on abnormal PRBC's.
• Most of the clinical manifestations of the disease are due to the lack of complement regulatory protein CD59.
Laboratory Findings

- Peripheral Smear: Macrocytic, normocytic or microcytic RBC’s
- Reticulocyte count: Mildly elevated
- Leukopenia and thrombocytopenia
- LDH is elevated
- Iron loss may amount to 20 mg/dl
- Urine hemosiderin
- Bone Marrow examination reveals erythroid hyperplasia

<table>
<thead>
<tr>
<th>Laboratory parameters</th>
<th>PNH case</th>
<th>Reference values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>5 g/dL</td>
<td>11.2-17.5 g/dL</td>
</tr>
<tr>
<td>Mean Corpuscular Volume</td>
<td>114.6 fL</td>
<td>80.0-100.0 fL</td>
</tr>
<tr>
<td>Reticulocyte count</td>
<td>22%</td>
<td>1-2%</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>3800 leukocytes/mmc</td>
<td>3980-10000 leukocytes/mmc</td>
</tr>
<tr>
<td>Haptoglobin</td>
<td>0.03 g/L</td>
<td>0.3-2 g/L</td>
</tr>
<tr>
<td>Ferritin</td>
<td>14 ng/mL</td>
<td>20-300 ng/mL</td>
</tr>
<tr>
<td>Leukocyte alkaline phosphatase score</td>
<td>0</td>
<td>10-100</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>4007 mmol/L</td>
<td>135-225 mmol/L</td>
</tr>
<tr>
<td>C3</td>
<td>86 mg/dL</td>
<td>90-180 mg/dL</td>
</tr>
</tbody>
</table>
Diagnostic Testing

• Flow cytometric techniques identify cell populations lacking GPI proteins (CD55 and CD 59) or GPI (FLAER)
Clinical Manifestations

Cerebral thrombosis
- Headache, nausea, vomiting

Retinal ven thrombosis
- Loss of vision

Abdominal vein thrombosis
- Budd-Chiari, splenic vein thrombosis, caput medusa, portal hypertension, esophageal varices and spasm, abdominal pain

Myocardial infarction
- Pulmonary embolism
- Pulmonary hypertension
- Dyspnea, hemoptysis

Renal failure
- Hypertension

Bone marrow failure
- Anemia, bleeding, infection

Erectile dysfunction

Cutaneous vein thrombosis
- Pyoderma gangrenosum

Deep venous thrombosis
- Pain, swelling, fever
Case Study

Case Study 1

- 28 year old male who presented to the hospital with generalized pain and fatigue. No chest pain, shortness of breath or neuro changes. No fevers.
- Labs on presentation Hgb 9.3 Retic 10.5% LDH 393 T.bili 3.4.
- During admission received 1 unit of blood on 9/26 for hgb 6.8 had an appropriate response 6.8 to 7.8 but 2 days later hgb 5.2
- Then received a exchange transfusion for acute chest syndrome on 9/28
- Which type of hemolytic anemia
- How will we treat
Case Study

Case #2

- 62 year old male who initially presented to his urologist with hematuria
- Labs on presentation: Hgb of 6.9
- Workup:
  - retic 10.26%  LDH 2371, haptoglobin was undetectable
  - T.bili 3.9 conjugated 1.0
  - Coombs test Anti IgG Negative. Anti C3b C3d Positive.
- Peripheral smear reviewed, showed agglutination of rbcs, atypical appearing lymphocytes.
- CMV  31277
- CT AP with enlarged spleen
- Bone marrow path
  - Slightly hypercellular (60-70%) marrow with trilineage hematopoiesis.
  - No obvious clonal plasmocytosis identified
  - No morphologic evidence of lymphoma/leukemia.
  - Markedly decreased iron stores in marrow.
- Which type of hemolytic anemia?
- How will we treat?


References


