

Unit 7: Hematology
Chapter 33 & 34
ONLINE CONTENT (2H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 16, 2026 at 0800. Please be sure to bring a copy to class on March 16, 2026.

Table 1	Iron Deficiency Anemia	Thalassemia	Cobalamin (Vitamin B₁₂) Deficiency	Folic Acid Deficiency
Etiology	Inadequate diet intake, malabsorption, blood loss, or hemolysis	Group of diseases involving inadequate production of normal Hgb, which decreases RBC production	Most common cause is pernicious anemia this happens when the body does not make intrinsic factor, which is needed to absorb vitamin B12. Occurs in GI sx or small bowel resection involving ileum. Others at risk are those with Crohn's disease, ileitis, celiacs disease, diverticula of the small intestine or chronic atrophic gastritis	Not enough folic acid in the body. Without it, RBCs cannot form & mature properly.
Clinical Manifestations	Early stages may not have any symptoms -Pallor most common -Glossitis (inflammation of the tongue) 2nd common -Cheilitis (inflammation of the lips) -Headache -Paresthesia -Burning sensation of tongue	Often asymptomatic in thalassemia minor Mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed skin color , and bone marrow hyperplasia	Sore, red, beefy, and shiny tongue; anorexia nausea, & vomiting; abdominal pain. Weakness, paresthesia of the feet and hands, reduced vibratory & position senses, ataxia, muscle weakness ad impaired cognition may take several weeks/years to develop	Similar to cobalamin deficiency/ may be attributed to other coexisting problems (cirrhosis, esophageal varices) GI include = stomatitis, cheilosis, dysphagia, flatulence, & diarrhea. Thiamine deficiency, which is often present with folate deficiency, can cause neurological problems
	Stool occult blood test, Endoscopy,	Labs (CBC, iron studies, genetic	Labs -RBCs may appear large	Serum Folate = low Normal serum

<p>Diagnostic Studies</p>	<p>colonoscopy, possible bone marrow biopsy & blood test Hgb/Hct MCV Reticulocytes Serum iron TIBC Transferrin Ferritin Bilirubin Serum B12 Folate</p>	<p>testing)</p>	<p>(macrocytic) & have abnormal shapes. Serum cobalamin levels are low. Normal serum folate levels & low cobalamin levels suggest megaloblastic anemia due to cobalamin deficiency. Pt with pernicious anemia have an increased risk for gastric cancer. Serum methylmalonic acid (MMA) (high in cobalamin deficiency and serum homocysteine help determine the cause of anemia</p>	<p>cobalamin level. Thiamine deficiency is often present and causes neurological symptoms.</p>
<p>Drug Therapy</p>	<p>Oral Ferrous sulfate or ferrous gluconate, IM or IV iron dextran, sodium ferrous gluconate, iron sucrose Nutrition therapy Packed RBC Infusion Vit c, vit b12, folic acid, copper, niacin, iron, Pantothenic acid (vit B5), Riboflavin (vit B2) Pyridoxine (vit B6), Vitamin C, Vitamin E Daily dose of iron should be 100-200 mg of elemental iron. Taken 3 or 4 daily doses with each table or capsule containing 60 to 70 mg of iron Iron is best absorbed in an acidic environment, avoid binding the iron with food, take 1 hour before meals vit c or OJ increased iron absorption</p>	<p>Blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron Drugs: oral deferasirox, or deferiprone, or IV or subcutaneous deferoxamine. Luspatercept-aamt may be given subcutaneously q 21 days (it improves Hgb levels & reduces transfusion needs and blocks inhibitors of late-stage RBC production</p>	<p>Without cobalamin administration the pt will die 1-3 years. Parenteral vitamin B12 or intranasal cyanocobalamin. 1000 mcg/day of cobalamin IM for 2 weeks, then weekly until Hgb normal, then monthly for life</p>	<p>. Tx with replacement therapy. 1 to 5 mg/day by mouth. Duration depends on reason for deficiency.</p>
<p>Nursing Management</p>	<p>Treat underlying problems, direct efforts toward</p>	<p>Thalassemia minor does not need tx b/c the body adapts to</p>	<p>Assess for neurologic problems that are not</p>	<p>Teach to eat foods high in folic acid. Foods include</p>

	replacing iron, teach pt which foods are good sources of iron	the reduction of normal Hgb	corrected by replacement therapy. Implement measures to reduce the risk of injury from the decreased sensitivity to head & pain. Protect from fall, burns, & trauma. Sometimes neuromuscular problems may not be reversible so may need physical therapy	Green leafy veg, enriched grain products & breakfast cereals, orange juice, peanuts, avocados
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Table 2	Anemia of Chronic Disease	Aplastic Anemia	Acute Anemia due to Blood Loss	Chronic Anemia due to Blood Loss
Etiology	Aka “anemia of inflammation” develops because of long-term illness or chronic inflammation. Under production of RBCs & mild shortening or RBC survival. Causes include: cancer, autoimmune & infectious disorders (HIV, Hepatitis, malaria), Hf, bleeding disorders	Due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target & destroy the patient’s own hematopoietic stem cells. Toxic injury to bone marrow stem cells or an inherited stem cell defect can also be the cause	Occurs with sudden bleeding. Trauma, sx, complications, & problems that disrupt vascular integrity. Two clinical concerns. A sudden reduction in the total blood volume can lead to hypovolemic shock, 2 nd if the acute loss is more gradual, the body maintains its blood volume by slowly increase the plasma volume	Long-term or repeated blood loss that gradually depletes iron stores
Clinical Manifestations	Fatigue, weakness, pallor, SOB, dizziness, headaches	Can manifest abruptly or over weeks/months. Varies from mild/severe. Fatigue, dyspnea as well as cardiovascular & cerebral responses may occur. Pt with neutropenia is susceptible to infection. Risk for septic shock & death. Thrombocytopenia	S/s are more important than the laboratory values – Pain , Internal bleeding may cause pain. Due to tissue distention, organ displacement, & nerve compression. Pain may be localized or referred to. In the case of retroperitoneal bleeding, the patient may not have	Fatigue, weakness, pallor, SOB, dizziness, headache

		can lead to bleeding	abdominal pain. Instead, there may be numbness & pain in a lower extremity from compression of the lateral cutaneous nerve. The major complication is shock	
Diagnostic Studies	High serum ferritin & increased iron sores distinguish it from other iron deficiency anemia. Normal folate & cobalamin blood levels	Laboratory studies confirm diagnosis. Because aplastic anemia affects all marrow elements, Hgb, WBC, & platelets values are decreased. Other RBC indices are generally normal. Reticulocyte count is low. Serum iron and total iron-binding capacity may be high as initial signs of decreased RBC production	When blood volume loss is sudden, plasma volume has not yet had a chance to increase. Lab data does not reflect RBC loss. Values may seem normal or high for 2-3 days. However, once the plasma volume is replaced, the RBC mass is less concentrated. Then, RBC, Hgb, & Hct levels are low & reflect the actual blood loss.	Hgb/Hct/MCV/serum iron/TIBC = low Reticulocytes normal or increased, Transferrin normal, Ferritin normal, bilirubin normal or decreased, serum B12 normal, folate normal
Drug Therapy	Correct underlying problem. If severe enough = blood transfusion. EPO therapy is used for anemia from renal disease & cancer & its therapies	Immunosuppressive therapy: Antihymocyte globulin (ATG), cyclosporine Elrombopag (Promacata): Oral thrombopoietin receptor agonist. It increases platelet count. High-dose cyclophosphamide, Alemtuzumab, Androgens (androgen used in select patients who do not respond to other tx, patients needing frequent transfusions give iron-binding agents to prevent iron overload	. Based on what's lost – Packed RBCs, whole blood, platelets, plasma, cryoprecipitate, IV Fluids: 0.9% NS, LR Colloids: Dextran, Hetastarch, albumin Body takes 2-5 das to make new RBC (via EPo) iron may be needed if stores are low (oral or IV iron if needed)	Iron supplements
	EPO therapy	Identifying and	Replace blood	Identify the source

Nursing Management	may be limited because of the increased risk for thromboembolism and death in some patients	removing the causative agent (when possible) & providing supportive care until the pancytopenia resolves.	volume (prevent shock), promote clotting, find and stop source of bleeding	and stop the bleeding
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Table 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	Results from hemolysis of RBCs from extrinsic factors. (physical destruction, antibody reaction, infectious agents & toxins) Increased RBC destruction	Iron overload disorder is characterized by increased intestinal iron absorption. Genetic defects are the most common cause. It may occur with diseases such as sideroblastic anemia and liver disease, and the chronic blood transfusions used to treat thalassemia and SCD.	Production & presence of increased number of RBCs. The increase in RBCs can be so great that blood circulation is impaired because of the increased blood viscosity (hyper viscosity) & volume (hypervolemia) 2 types: Polycythemia or polycythemia vera, & secondary polycythemia. Their causes and pathogenesis differ, although their complications and manifestations are similar
Clinical Manifestations	Fatigue, hypotension, pallor , high cardiac outputs leading to congestive heart failure Jaundice Dark urine General anemia symptoms	Do not normally develop until after age 40 in men and after 50 for women. Fatigue, arthralgia, impotence, abdominal pain, & weight loss. Later, the excess iron deposits in the liver, pancreas, heart, joints, and endocrine glands cause diabetes, skin pigment changes (bronzing), heart problems (cardiomyopathy), arthritis, and testicular atrophy. May be enlarged liver and spleen and skin pigmentation changes.	Primary- Increased product not only of RBCs but also WBCs and platelets. Splenomegaly & hepatomegaly are common. Secondary- hypoxia or hypoxia independent. Hypoxia driven, Circulatory manifestations occur due to hypertension caused by hypervolemia and hyperviscosity. -headache -vertigo -dizziness -tinnitus -visual changes Generalized itching is often exacerbated by hot baths, which may be a striking symptom.

			<p>-Paresthesia and erythromelalgia (painful burning and redness of hands and feet)</p> <p>-Angina</p> <p>-Intermittent claudication</p> <p>-Thrombophlebitis</p>
Diagnostic Studies	<p>Hgb/Hct= LOW</p> <p>MCV=Normal</p> <p>Reticulocytes = Increased</p> <p>Serum iron = Normal or increased</p> <p>TIBC = Normal</p> <p>Transferrin = Normal</p> <p>Ferritin = Normal or increased</p> <p>Bilirubin = Increased</p> <p>Serum b12= normal</p> <p>Folate = normal</p>	<p>High serum iron, TIBC and serum ferritin. Testing for known genetic mutations confirms the diagnosis. MRI can measure liver and cardiac iron. Liver biopsy can quantify the amount of iron and establish the degree of organ damage</p>	<p>High Hgb</p> <p>High Hct</p> <p>High RBC mass</p> <p>Bone marrow examination showing hypercellularity of RBCs, WBCs, and platelets; and presence of JAK2 V617F or JAK2 exon 12 mutation. Other studies show LOW EPO level, high wbc with basophilia and neutrophilia, high platelet count and platelet dysfunction and normal or high leukocyte alkaline phosphate, uric acid, and cobalamin levels</p>
Drug Therapy	<p>Folate placement for chronic hemolytic anemia, to suppress RBC destruction, immunosuppressive agents may be used (glucocorticoids or rituximab) Severe: thrombocytopenia, & AKI additional supplements (cyclosporine) may be used. Plasma exchange & eculizumab, a monoclonal antibody to complement protein C5 are options</p>	<p>Removal of 500 ml of blood each week until the iron stores are depleted. Then blood is removed less often to maintain iron levels within normal limits. Iron-chelating drugs may be used. Deferoxamine chelates and removes iron via the kidneys. It is given IV or Subcutaneously. Deferasirox ad deferiprone are oral drugs. Diet changes include avoiding vitamin C, and iron supplements, uncooked seafood, and iron-rich foods</p>	<p>Treatment aims to reduce blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment The aim of phlebotomy is to reduce the hct and keep it less than 45%. At the time of dx 300-500 ml of blood may be removed every few days until the hct is reduced to acceptable levels. Hydration therapy Low dose ASA can prevent clotting Myelosuppressive agents -hydroxyurea -busulfan Ruxolitinib (which inhibits expression of the JAK2 mutation, is given to those who do not respond to hydroxyurea. A-Interferon – 2b and pegylated IFN alfa- 2a are options for women of childbearing age or those with intractable itching</p>

<p>Nursing Management</p>	<p>General supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs. Because a hemolytic crisis is a potential consequence, be ready to institute appropriate emergency therapy. This includes aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by Hgb clogging the kidney tubules and subsequent shock. Supportive care may involve corticosteroids and blood products or removing the spleen</p>	<p>Manage problems from organ involvement (diabetes, HF) with the usual treatment of these problems. The most common causes of death are cirrhosis, liver failure, liver cancer, and HF. With early dx and tx, life expectancy is normal.</p>	<p>Assess patients' nutrition status/ Inadequate food intake can result from GI symptoms of fullness, pain, and dyspepsia. Begin activities and drug therapy to decrease thrombus formation. Requires ongoing evaluation.</p>
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In order to receive full credit (2H class time) for this assignment, it must be completed in its entirety by the due date/time assigned. Any assignment not completed in its entirety by the due date and time will result in missed class time and must be completed by the end of the semester to pass the course.