

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

**Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 18, 2024 at 0800. Please be sure to bring a copy to class on March 18, 2024.**

Table 1	<b>Iron Deficiency Anemia</b>	<b>Thalassemia</b>	<b>Cobalamin (Vitamin B<sub>12</sub>) Deficiency</b>	<b>Folic Acid Deficiency</b>
<b>Etiology</b>	-May develop from inadequate diet intake, malabsorption, blood loss, or hemolysis	-A group of diseases involving inadequate production of normal HGB, which decreases RBC production -Due to the absent or reduced globulin protein	-Most common cause is pernicious anemia. -Caused by an absence of intrinsic factor. Begins in middle age or later	-Can cause megaloblastic anemia
<b>Clinical Manifestations</b>	Early: No symptoms Chronic: Pallor, glossitis, cheilitis, headache, paresthesia, and burning sensation of the tongue	-Often asymptomatic -Microcytosis, hypochromia, mild splenomegaly, bronzed skin color, bone marrow hyperplasia	-sore, beefy, shiny tongue, anorexia, nausea, vomiting, abdominal pain, weakness, paresthesia of the feet and hands, reduced vibratory and position sense, ataxia, muscle weakness, impaired cognition	-Stomatitis, cheilosis, dysphagia, flatulence, diarrhea, thiamine deficiency
<b>Diagnostic Studies</b>	-Low HGB/HCT -Low MCV -Low serum Iron -None or low bilirubin -colonoscopy or endoscopy, bone marrow biopsy	-Low HGB/HCT -No or low MCV -increased serum iron -increased bilirubin -decreased folate	-RBCs- large and have abnormal shapes -Serum cobalamin levels are low, normal serum folate levels	-Serum folate is low, a normal cobalamin
<b>Drug Therapy</b>	- Ferrous sulfate or ferrous gluconate - Iv: iron dextran, sodium ferrous gluconate,	-Oral deferasirox, deferiprone, or IV deferoxamine -Rebloyzl every 21 days	-Parenteral vitamin B12 or intranasal cyanocobalamin	-Replacement therapy of folic acid

	iron sucrose			
<b>Nursing Management</b>	<ul style="list-style-type: none"> <li>-Identify and treat underlying cause</li> <li>-packed RBC infusion</li> <li>-Teach pts good foods that contain iron</li> </ul>	<ul style="list-style-type: none"> <li>-Minor- does not need treatment</li> <li>-Major- blood transfusions, may need splenectomy</li> <li>-Monitor heart, liver, and lung function.</li> <li>-Hematopoietic stem cell transplantation is the only cure</li> </ul>	<ul style="list-style-type: none"> <li>-Assess for neurologic problems</li> <li>-Protect the patient from falls, burns, and trauma</li> <li>-May need physical therapy</li> </ul>	<ul style="list-style-type: none"> <li>-Teach patients to eat foods high in folic acids</li> </ul>

Table 2	<b>Anemia of Chronic Disease</b>	<b>Aplastic Anemia</b>	<b>Acute Anemia due to Blood Loss</b>	<b>Chronic Anemia due to Blood Loss</b>
<b>Etiology</b>	<ul style="list-style-type: none"> <li>-Usually develops after 1-2 months of disease activity</li> <li>-Chronic: is associated with an underproduction of RBC and mild shortening of RBC survival</li> </ul>	<ul style="list-style-type: none"> <li>-70% of aplastic anemia are due to autoimmune activity by autoreactive T lymphocytes.</li> </ul>	<ul style="list-style-type: none"> <li>-Causes of acute blood loss include trauma, surgery complications, and problems that disrupt vascular integrity.</li> </ul>	<ul style="list-style-type: none"> <li>-Causes are similar to those of iron deficiency anemia. The effects are usually due to depleted iron stores.</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>-Mild: palpitations, exertional dyspnea</li> <li>-Severe: Increased HR, Increased pulse pressure, systolic murmurs, anorexia, hepatomegaly, weight loss, glossitis, bone pain headache, tachypnea</li> </ul>	<ul style="list-style-type: none"> <li>-Fatigue, dyspnea, cardiovascular and cerebral responses may occur, risk of septic shock and death</li> </ul>	<ul style="list-style-type: none"> <li>-2000mL lost- central venous pressure and cardiac output below normal at rest, air hunger, rapid, thready pulse, and cold clammy skin</li> <li>-2500mL lost- Shock, lactic acidosis, death</li> </ul>	<ul style="list-style-type: none"> <li>-2000mL lost- central venous pressure and cardiac output below normal at rest, air hunger, rapid, thready pulse, and cold clammy skin</li> <li>-2500mL lost- Shock, lactic acidosis, death</li> </ul>
<b>Diagnostic Studies</b>		<p>Hgb, WBC and platelet values are decreased. Bone marrow biopsy, aspiration.</p>	<ul style="list-style-type: none"> <li>-Lab values don't reflect the RBC lost. Once replaced the RBC mass is less</li> </ul>	<ul style="list-style-type: none"> <li>-RBC, Hgb, HCT levels are low</li> </ul>

	-High serum ferritin, increased iron		concentrated	
<b>Drug Therapy</b>	-Blood transfusions, EPO therapy	-HSCT is critical, or Immunosuppressive therapies	-Blood transfusions, with volume replacement fluids if in an emergency	-May need an iron supplement
<b>Nursing Management</b>	-Find the underlying cause, blood transfusions if severe	-Identifying and removing the causative agents, prevent complications from infection and bleeding	- Initially concerned with replacing blood volume to prevent shock, promoting coagulation to prevent further bleeding, and finding the source of the bleeding and stopping the blood loss	-Identifying the source and stopping the bleeding

Table 3	<b>Acquired Hemolytic Anemia</b>	<b>Hemochromatosis</b>	<b>Polycythemia</b>
<b>Etiology</b>	-Results from hemolysis of RBCs from extrinsic factors. These factors are physical destruction, antibody reaction, infectious agents, and toxins	-Iron overload disorder characterized by increased intestinal iron absorption. May occur with disease such as sideroblastic anemia and liver disease.	-There are 2 types: primary and secondary polycythemia. -Primary: It involves increased production not only of RBC's but also WBCs and platelets -Secondary: can be either hypoxia driven or hypoxia independent
<b>Clinical Manifestations</b>	-Mild: palpitations, exertional dyspnea -Severe: Increased HR, Increased pulse pressure, systolic murmurs, anorexia, hepatomegaly, weight loss, glossitis, bone pain headache, tachypnea	-symptoms usually don't appear until after age 40 years in men and 50 years in women. Fatigue, arthralgia, impotence, abdominal pain, weight loss -Later: excess iron accumulates in the liver, pancreas, heart, joints and endocrine cause diabetes, skin pigment changes, heart problems, arthritis, and testicular atrophy.	-Headache, vertigo, dizziness, tinnitus, and visual changes, generalized itching, paresthesia, erythromelalgia, angina, HF, intermittent claudication, thrombophlebitis
<b>Diagnostic Studies</b>	-Decreased	-High serum iron, TIBC, and serum ferritin. -Testing for genetic mutations, MRI, liver biopsy	-High Hgb, HCT, and RBC mass -Bone marrow examination showing hypercellularity or

	Hgb/HCT		RBCS, WBCS and platelets -High WBC count with basophilia and neutrophilia -High platelet count and platelet dysfunction
<b>Drug Therapy</b>	-Folate replacement such as glucocorticoids	-Iron-chelating drugs, deferoxamine or oral drugs: deferasirox and deferiprone	Myelosuppressive agents, Ruxolitinib
<b>Nursing Management</b>	-Aggressive hydration and electrolyte replacement, supportive care	-Teach pt about avoiding vitamin C, and iron supplements, uncooked seafood, and iron rich foods	Assess intake and output during hydration therapy, assess the patient's nutrition status

***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.***