Unit 7: Hematology
Chapter 29 & 30
ONLINE CONTENT (1.5 H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 20, 2023 at 0800. Please be sure to bring a copy to class on March 20, 2023.

Table 1	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	-May develop because inadequate dietary intake, malabsorption, blood loss, or hemolysis. -Most common nutritional deficiency in the world	-A group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production. -Due to an absent or reduced globulin protein. -Commonly found in members of ethnic groups whose origins are near the Mediterranean Sea and equatorial or near-equatorial regions of Southern Asia, the Middle East, India, Pakistan, China, Southern Russia, and Africa.	-most common cause of cobalamin deficiency is pernicious anemia. -caused by an absence of intrinsic factor. -begins in middle age or > 40yrs old -60 years old is the most common age -Without IF cobalamin will not be absorbed -familial predisposition for pernicious anemia, evaluate patients who have a positive family history of pernicious anemia	-folate deficiency -causes megaloblastic anemia -needed for DNA synthesis leading to RBC formation and maturation Common causes: -chronic alcoholism -malabsorption syndromes -dietary deficiency -chronic hemodialysis
Clinical Manifestations	In the early course of iron-deficiency anemia, the patient may not have any symptoms. As the disease becomes chronic general manifestations include pallor, glossitis (tongue inflammation), and cheilitis (inflammation of the lips). The patient may report headaches, paresthesia, and a burning sensation of the tongue, all of which are caused by a lack of iron in the tissues.	<u>Thalassemia Minor:</u> -often asymptomatic -mild to moderate anemia with microcytosis(small cells) and hypochromia (pale cells) -mild splenomegaly -bronzed color of skin -bone marrow hyperplasia <u>Thalassemia Major:</u> -physical and mental growth slowed -pale -growth/developmental deficits -other general anemia symptoms -Jaundice -Pronounced splenomegaly -Hepatomegaly -Cardiomyopathy	-General manifestations of anemia r/t cobalamin deficiency develop d/t hypoxia. -GI manifestations: - sore/red/beefy/shiny tongue -anorexia -nausea -vomiting -abdominal pain Neuromuscular manifestations include: -weakness -paresthesia of the feet and hands -reduced vibratory and position senses -ataxia -muscle weakness -impaired thought process ranging from confusion to dementia	-Symptoms may be attributed to other coexisting problems such as cirrhosis/esophageal varices GI Issues: -stomatitis -cheilosis -dysphagia -flatulence -diarrhea -neurologic symptoms
Diagnostic	Labs results- -Hgb/Hct= decreased -MCV= decreased	Labs results- Hgb/Hct= decreased MCV=normal or	Lab results- Hgb/Hct= decrease MCV=increased	Labs results- Hgb/Hct= decreased MCV=increased

Studies	-Reticulocytes= N or slightly increased or decreased -Serum Iron= decreased -TIBC= increased -Transferrin= N or decreased -Ferritin= decreased -Bilirubin= N or decreased -Serum B12= N -Folate= N Stool Occult Blood Test Endoscopy/Colonoscopy Bone Marrow Biopsy	decreased Reticulocytes=increased Serum Iron= increased TIBC= decreased Transferrin=decreased Ferritin= normal or increased Bilirubin= increased Serum B12= Normal Folate= decreased	Reticulocytes=normal or decreased Serum Iron= normal or increased TIBC= normal Transferrin=slight increase Ferritin= increased Bilirubin= normal or slight increase Serum B12= decreased Folate= normal	Reticulocytes=normal of decreased Serum Iron= normal or increased TIBC= normal Transferrin=slight increase Ferritin=increased Bilirubin=normal or slight increase Serum B12= N Folate= decreased
Drug Therapy	*Oral Iron *150-200mg daily *Take an hour before meals with vitamin C *GI side effects: heartburn/constipation/diarrhea *Pt stay upright for 30min after taking oral form *Start on stool softeners and laxatives if needed	-No specific drug or diet therapies are effective in treating -Thalassemia minor does not need treatment because the body adapts to the reduction of normal hemoglobin -Thalassemia major is managed with blood transfusions in conjunction with chelating agents that bind to iron -Chelating agents reduce iron overloading that occurs with chronic transfusion therapy -Oral deferasirox(Exjade, JadeNu) Deferiprone (Ferriprox) -IV/ subcutaneous deferoxamine (Desferal) -Ascorbic Acid supplements -Folic Acid (if evidence of hemolysis) -Zinc supplements	-Parenteral vitamin B12 or intranasal cyanocobalamin Typical treatment: -1,000 mcg/day of cobalamin IM for 2 weeks then weekly until the hemoglobin is normal then monthly for life.	Replacement Therapy--1mg/day by mouth Malabsorption/chronic alcoholism -5mg/day
Nursing Management	-Identify and treat underlying cause -Drug therapy -Oral: Ferrous sulfate or ferrous gluconate -IM/IV: iron dextran, sodium ferrous gluconate, iron sucrose -Transfusion of packed RB	-monitor hepatic/heart/lung function -give meds as ordered	-Implement measures to reduce the risk for injury from the decreased sensitivity to heat & pain related to neurologic impairment. -Protect patient from falling/burns/trauma.	-Teach patients to eat foods high in folic acid - green leafy vegetables. Enriched grain, products and breakfast cereals, orange juice, peanuts, avocado

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	-Associated w/ an underproduction of RBCs and mild shortening of RBC survival	-Caused by autoimmune activity by autoreactive T lymphocytes -The cytotoxic T cells target and destroy the patient's hematopoietic stem cells	-Acute blood loss occurs because of sudden hemorrhage -Causes of acute blood loss include: -trauma -complications of surgery	-Effects of chronic blood loss are usually r/t the depletion of iron stores and considered an iron deficiency anemia.

			-conditions/diseases that disrupt vascular integrity -hypovolemic shock & a decrease in RBC's caring O2 are clinical concerns	
Clinical Manifestations	-Develops after 1 to 2 months of disease activity and has an immune basis -Interkeukin-6 causes an increased uptake and retention of iron within macrophages -erythropoietin production is decreased	Aplastic anemia can manifest abruptly over days or insidiously over weeks to months. -Varies from mild to severe. General manifestations: -Fatigue -dyspnea -cardiovascular -cerebral responses -low neutrophil count make pt susceptible to infection and at risk for shock and death.	-10%(500mL)= none or rare vasovagal syncope -20%(1,000mL)= No detachable s/s at rest. Tachycardia w/ exercise and slight postural hypotension -30%(1,500mL)= normal supine blood pressure and pulse at rest. Postural hypotension and tachycardia w/ exercise -40%(2,000mL)= BP, central venous pressure, and cardiac output below normal at rest; air hunger; rapid/thready pulse, and clod/clammy skin -50%(2,500mL)= shock, lactic acidosis, & potential death	The sources of chronic blood loss are similar to those of iron deficiency anemia such as: -bleeding -ulcers -hemorrhoids -menstrual & postmenopausal blood loss
Diagnostic Studies	Lab results- Hgb/Hct= decrease MCV=normal or decreased Reticulocytes=normal or decreased Serum Iron= normal or decreased TIBC= decreased Transferrin=normal or decreased Ferritin= normal or increased Bilirubin= normal Serum B12= normal Folate= normal	Hgb/Hct- decreased MCV- normal or increased Reticulocytes- decreased Serum Iron- normal or increased TIBC-normal or increased Transferrin- normal Ferritin- normal Bilirubin- normal Serum B12- normal Folate- normal	Lab results- Hgb/Hct= decrease MCV=normal or decreased Reticulocytes=normal or increased Serum Iron= normal TIBC= normal Transferrin=normal Ferritin= normal Bilirubin= normal Serum B12= normal Folate= normal	Hgb/Hct- decreased MCV- decreased Reticulocytes- normal or increased Serum Iron- decreased TIBC-decreased Transferrin- normal Ferritin- normal Bilirubin- normal or decreased Serum B12- normal Folate- normal
Drug Therapy	-Correct the underlying disorder. -anemia is severe blood transfusions may be needed, but they are not recommended for long term treatment.	-Management of aplastic anemia is based on identifying and removing the causative agent when possible and providing supportive care until the pancytopenia reverses	-IV fluids for emergencies: -dextran -hetastarch -albumin -crystalloid electrolyte solutions (ex. lactated ringers solution) -Blood transfusions(Packed	-Supplemental iron (may be needed)

			RBCs) -May give platelets /plasma/cryoprecipitate -supplemental iron (oral or parenteral)	
Nursing Management	-Administer blood transfusions	-Mostly is directed towards preventing complications from infection and hemorrhage	-For postoperative pt carefully monitor the blood loss from various drainage tubes/dressings and implement appropriate actions -giving blood products	Management of chronic blood loss anemia involves: -identifying the source -stopping the bleeding

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	-Results from hemolysis of RBCs from extrinsic factors Factors includes: -physical destruction -antibody reactions -infectious agents -toxins	-Iron overload disorder -genetic defect is most common cause -also caused by sideroblastic anemia/liver disease/and chronic blood transfusions	-production & presence of increased number of RBCs -Increase in RBCs can be so great that blood circulation is impaired d/t increased blood viscosity and volume -Two types: -primary polycythemia -secondary polycythemia.
Clinical Manifestations	-dyspnea -angina -tachycardia -malaise -dizziness	-s/s develop after age 40 in men and after age 50 in women -early s/s are nonspecific & include: -fatigue -arthralgia -impotence -abdominal pain -weight loss -Later excess iron accumulates in liver and causes liver enlargement and cirrhosis. -diabetes -skin pigment changes (bronzing) -heart problems -arthritis -testicular atrophy	-Headache -vertigo -dizziness -tinnitus -visual changes -Generalized pruritus after a hot bath (striking symptom) -burning and redness of hands/feet
Diagnostic Studies	Hgb/Hct- decreased MCV- normal or increased Reticulocytes- increased Serum Iron- normal or increased TIBC-normal or decreased Transferrin- normal Ferritin- normal or increased Bilirubin- increased Serum B12- normal Folate- normal	Lab Values: -high serum iron -high TIBC -high serum ferritin -Testing for know genetic mutations confirms the diagnosis -Liver biopsy can establish the degree of organ damage	-high hemoglobin and RBC count with microcytosis -low to normal EPO level (Secondary high) - high WBC count with basophilia and neutrophilia -high platelet count and platelet dysfunction -high leukocyte alkaline phosphatase, uric acid and cobalamin levels -high histamine levels -Bone marrow examination in polycythemia (shows hypercellularity of RBCs, WBCs, and platelets)
Drug Therapy	The patient with chronic hemolytic anemia may need: -folate replacement	-removing 500mL of blood each week for 2-3 years until the iron stores in the body are depleted	-Low dose aspirin is used to prevent clotting. -Avoid iron supplements

	-immunosuppressive agents (glucocorticoids or rituximab) may be used in order to suppress the destruction of RBCs	-iron chelating agents (Deferoxamine, IV or subcutaneously) -Deferasirox and Deferiprone(oral agents)	-Hydration therapy can reduce the blood's viscosity -Anagrelide can reduce the platelet count and inhibit platelet aggregation -Pegylated interferons are given to women of childbearing age or those with intractable pruritus -Allopurinol may reduce the number of acute gouty attacks.
Nursing Management	-Treatment and management of acquired hemolytic anemias involve: -general supportive care until the causative agent can be eliminated or at least made less injurious to the RBC's.	-Teach pt to avoid vitamin C/iron supplements/ uncooked seafood/ and iron rich foods.	Treatment =reducing blood volume/viscosity and bone marrow activity -Phlebotomy is the mainstay of treatment