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	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	Inadequate diet intake, malabsorption, blood loss, hemolysis, people with higher iron needs (menstruating/pregnant women).	Absent or reduced globulin protein. Alpha-Globin chains are absent or reduced in alpha-thalassemia, beta-Globin chains are absent or reduced in beta-thalassemia. Hemolysis occurs as mononuclear phagocytes in the marrow destroy most erythroblasts. Those released into blood are rapidly destroyed by macrophages in the spleen.	Pernicious anemia (most common cause). Absence of intrinsic factor- without intrinsic factor the body cannot absorb cobalamin. GI surgery, small bowel resection involving the ileum, Crohn disease, ileitis, celiac disease, diverticular of the small intestine, chronic atrophic gastritis. Loss of IF-secreting gastric mucosal cells or impaired absorption of cobalamin in the distal ileum. Also occurs with excess alcohol or hot tea ingestion, smoking, long-term users of H2-histamine receptor blockers and proton pump inhibitors, and those who are strict vegetarians. Familial predisposition for pernicious anemia.	Chronic alcohol use, chronic hemodialysis, diet deficiency, drugs interfering with absorption or use of folic acid, increased requirement (pregnancy), malabsorption syndromes (celiac disease, Crohn disease, small bowel resection)
Clinical Manifestations	Early- patient may not have symptoms. As disease becomes chronic, any of general manifestations of anemia may develop. Pallor (most common), glossitis, cheilitis, headache, paresthesias, burning sensation of tongue. Anemia manifestations: tissue hypoxia, palpitations,	Minor- patients often asymptomatic; have mild to moderate anemia with microcytosis and hypochromia, mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Major- life-threatening; symptoms develop in childhood by age 2 and can cause	Tissue hypoxia; sore, red, beefy, and shiny tongue; anorexia; nausea and vomiting; abdominal pain. Weakness, paresthesias of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired cognition. Manifestations may take several months or	Similar to cobalamin deficiency. Develops insidiously. Stomatitis, cheilosis, dysphagia, flatulence, diarrhea. When concurrent with thiamine deficiency, neurologic problems can occur.

	dyspnea, mild fatigue, pallor, jaundice, itching, hemolysis.	growth and development deficits. Pale, general symptoms of anemia, jaundice, pronounced splenomegaly, hepatomegaly, cardiomyopathy, chronic bone marrow hyperplasia and expansion of marrow space, thickening of the cranium and maxillary cavity, thrombocytosis after spleen dysfunction and/or removal. Cardiac complications from iron overload, lung disease, and hypertension contributes to early death. Endocrine problems (diabetes, growth retardation, hypogonadism), osteoporosis, pulmonary hypertension, and thrombosis may occur.	years to develop.	
Diagnostic Studies	Laboratory values (Hgb, Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, folate), stool occult blood test, endoscopy, colonoscopy, bone marrow biopsy.	Laboratory values (Hgb, Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, folate, RBC count)	Laboratory values (Hgb, Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, folate), serum test for anti-IF antibodies, upper GI endoscopy and biopsy of gastric mucosa, serum methylmalonic acid, serum homocysteine	Laboratory values (Hgb, Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, folate)
Drug Therapy	Oral: ferrous sulfate or ferrous gluconate IM or IV: iron dextran, sodium ferrous gluconate, iron sucrose	Minor: no treatment Major: blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron; Reblozyl (improves Hgb levels and reduces transfusion needs)	Parenteral vitamin B12 or intranasal cyanocobalamin Normal treatment schedule: 1000 mcg/day of cobalamin IM for 2 weeks, then weekly until Hgb normal, then monthly for life	Folic acid replacement therapy: usual dosage 1-5 mg/day by mouth. Duration of treatment depends on reason for deficiency.
Nursing Management	Treat underlying problem causing iron loss, reduced intake, or poor iron absorption. Replace iron needed.	Monitor blood transfusions: monitor liver, heart, and lung function with splenectomy and	Assess for neurologic problems not corrected by replacement therapy, implement measures to reduce	Teach patient to eat foods high in folic acid. If patient has fatigue, encourage alternate rest and

	Teach patient which foods are good sources of iron, and if adequate already, educate patient they may need oral or occasionally IV iron supplements. Patient may need packed RBC transfusion if acute blood loss is the cause.	provide treatment as needed; monitor patient condition and notify provider if patient is thalassemia minor develops to thalassemia major.	risk for injury from decreased sensitivity to heat and pain, protect patient from falling, burns, and trauma Refer patient to physical therapy if neuromuscular problems not reversible	activity periods. Help patient prioritize activities according to energy level. Arrange activities to reduce competition for O2 supply to vital functions. Aid with regular activities, monitor patient's cardiorespiratory response to activity.

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Cancer, autoimmune and infectious disorders, HF, or chronic inflammation. Bleeding episodes can contribute.	Autoimmune activity by autoreactive T lymphocytes, toxic injury to bone marrow stem cells, inherited stem cell defect.	Trauma, surgery complications, problems that disrupt vascular integrity	Similar to iron deficiency anemia (bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss)
Clinical Manifestations	Reduced RBC life span, decreased erythropoietin production, ineffective bone marrow response to EPO. Tiredness, shortness of breath, pallor, diaphoresis, headaches, weakness, dizziness.	Abrupt or insidious onset. General manifestations of anemia (fatigue, dyspnea), cardiovascular and cerebral responses, infection (in neutropenic patients), thrombocytopenia can lead to bleeding (petechiae, bruising, nosebleeds).	10%, 500 mL: none or rare vasovagal syncope 20%, 1000 mL: no detectable signs or symptoms at rest. Increased HR with exercise and slight postural hypotension 30%, 1500 mL: normal supine BP and pulse at rest. Postural hypotension and increased HR with exercise 40%, 2000 mL: BP, central venous pressure, and cardiac output below normal at rest; air hunger; rapid, thready pulse and cold, clammy skin 50%, 2500 mL: shock, lactic acidosis, potential death	General manifestations of anemia may develop. Pallor (most common), glossitis, cheilitis, headache, paresthesias, burning sensation of tongue. Anemia manifestations: tissue hypoxia, palpitations, dyspnea, mild fatigue, pallor, jaundice, itching, hemolysis.
Diagnostic Studies	Laboratory values (MCV, serum ferritin, serum iron level, Hgb, reticulocyte count)	Laboratory values (Hgb, WBC, platelet values, reticulocyte count, serum iron, TIBC) Bone marrow biopsy, aspiration, pathologic examination	Lab values may seem normal/high for 2-3 days, do not reflect RBC loss. Once plasma volume replaced, RBC mass is less concentrated. Then, RBC, Hgb, Hct levels low and reflect actual blood loss.	Laboratory values (Hgb, Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, folate), stool occult blood test, endoscopy, colonoscopy, bone marrow biopsy.

Drug Therapy	Severe anemia: blood transfusions possibly needed. EPO therapy (limited use) for anemia from renal disease and cancer and its therapies.	HSCT, immunosuppressive therapy with antithymocyte globulin and cyclosporine, iron-binding agent, Eltrombopag, high-dose cyclophosphamide, alemtuzumab, androgens	Blood transfusions (packed RBCs, whole blood, platelets, plasma, cryoprecipitate), IV fluids (NS, LR), colloids (dextran, hetastarch, albumin), iron supplements	Iron supplements
Nursing Management	Monitor patient's symptoms, administer blood transfusions/medications as needed, provide emotional support, refer patient to community resources, assist patient with activities.	Provide supportive care, prevent complications from infection and bleeding, monitor patient symptoms and lab values, medication administration, provide patient comfort.	Carefully monitor blood loss from various drainage tubes and dressings and implement appropriate actions. Administer medications/blood/IV fluids as needed, monitor patient's symptoms, provide emotional support.	Assist with identifying source of blood loss and stopping bleeding. Encourage patient to eat foods high in iron to assist with increasing iron levels. Obtain patient's past medical and surgical histories to determine any conditions/recent surgeries that could be causing anemia.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	External factors cause damage to RBCs. Macrophages (mainly those in spleen, liver, and bone marrow) destroy RBCs that are old, defective, or moderately damaged at a faster rate than production of RBCs.	Genetic defect (most common), sideroblastic anemia, liver disease, chronic blood transfusions to treat thalassemia and SCD.	Primary polycythemia: 1 or more DNA mutations of a single hematopoietic stem cell, familial disposition Secondary polycythemia: - Hypoxia-driven: high altitude, lung disease, cardiovascular disease, alveolar hypoventilation, defective O2 transport, tissue hypoxia - Hypoxia-independent: cancer or benign tumor tissue
Clinical Manifestations	General manifestations of anemia, along with jaundice, splenomegaly, hepatomegaly.	Usually do not develop until after age 40 in men and 50 in women. Early- nonspecific, includes fatigue, arthralgia, impotence, abdominal pain, weight loss Later- liver enlargement, cirrhosis Excess iron causes diabetes, skin pigment changes (bronzing), heart problems,	Circulatory manifestations (often first manifestations): headache, vertigo, dizziness, tinnitus, visual changes. Generalized itching, paresthesias, erythromelalgia, angina, HF, intermittent claudication, thrombophlebitis. Petechiae, bruising, nosebleeds, GI bleeding.

		arthritis, and testicular atrophy Splenomegaly	Hepatomegaly, splenomegaly, pain from peptic ulcers, plethora, gout
Diagnostic Studies	Reticulocyte count, AST, bilirubin levels, urinalysis, bone marrow biopsy, haptoglobin, LDH	Serum iron levels, TIBC, serum ferritin Testing for known genetic mutations confirms diagnosis MRI, liver biopsy	Hgb, Hct, RBC mass. Bone marrow examination: hypercellularity of RBCs, WBCs, platelets. Presence of JAK2 V617F or JAK2 exon 12 mutation EPO level, WBC count, platelet count, leukocyte alkaline phosphatase, uric acid, cobalamin levels.
Drug Therapy	Corticosteroids, folic acid, rituximab, IVIG	Iron-chelating drugs (deferoxamine, deferasirox, deferiprone) Drugs used to manage other conditions caused by hemochromatosis (diabetes, HF)	Hydration therapy, low-dose aspirin, myelosuppressive agents, ruxolitinib, alpha-interferon-2b and pegylated IFN alfa-2a
Nursing Management	Manage patient's fatigue and other symptoms, medication administration, monitor patient's lab values, educate patient about condition, maintain renal function.	Encourage patient to make diet changes to avoid vitamin C and iron supplements, uncooked seafood, and iron-rich foods. Administer medications for hemochromatosis and other conditions caused by this, monitor patient symptoms, educate patient to report worsening symptoms, obtain past medical history and family history.	Assist with/perform phlebotomy, assess I&O during hydration therapy, give myelosuppressive agents as ordered, observe patient for worsening symptoms/side effects of medications, educate patient about drug side effects, assess patient's nutrition status, begin activities and any drug therapy to decrease thrombus formation, assess patient for complications with each encounter.

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.