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 from inadequate dietary intake, malabsorption, blood loss, or hemolysis -Seen in the young, poor diets, and women in reproductive years	-Group of diseases involving inadequate production of Hgb, which decreased RBC production -absent/ reduced globulin protein -Genetic link	-most common cause is pernicious anemia caused by absent intrinsic factor -lack of IF = cobalamin cannot be absorbed -can occur from GI surgery, Crohn's disease, ileitis, celiac disease, diverticula -excess alcohol or hot tea ingestion, smoking, long-term PPI users, and strict vegetarians	-causes megaloblastic anemia -folic acid needed for DNA synthesis
Clinical Manifestations	-May be asymptomatic -Pallor, glossitis, cheilitis, headache, paresthesia, burning sensation of tongue	-often asymptomatic -life threatening disease with slowed growth (mental and physical) -jaundice from hemolysis of RBC's -Splenomegaly	-sore red beefy and shiny tongue, anorexia, N/V, ABD pain, weakness, paresthesia of feet and hands, ataxia, impaired thought process (confusion to dementia) -may take months to develop	-same as cobalamin deficiency -stomatitis, cheilosis, dysphagia, flatulence, diarrhea -thiamine deficiency
Diagnostic Studies	-Low Hgb/Hct, low MCV, low serum iron, low, transferrin, low bilirubin, no folate -stool occult blood test, endoscopy or colonoscopy for GI bleed, bone marrow BX	-Genetic testing -symptoms present around 2 years of age	-large RBC's with abnormal shape = lysis because of fragile membrane -increased risk for GI cancer -increased MCV, decreased Hgb/Hct, increased serum iron, DECREASED SERUM B12	-Low serum folate level and normal serum cobalamin level
Drug Therapy	-Oral iron (take 1 hr before meal, taking	-transfusions -chelating agents	-parenteral vitamin b12	-Treat with replacement

	with orange juice enhances absorption) -parenteral iron IM or IV -may stain skin	that bind to iron (oral deferasirox, deferiprone, IV or subQ deferoxamine	(cyanocobalamin, hydroxocobalamin) or intranasal → without cobalamin administration patient will die in 1-3 years! - 1000mcg/day of cobalamin IM for 2 weeks then weekly then monthly for life	therapy: 1mg/day PO folic acid (up to 5mg for alcoholism and malabsorption)
Nursing Management	-Teach pts to increased dietary iron intake -may need transfusion of PRBCs	-prepare/ recover possible splenectomy -hematopoietic stem cell transplant only cure but the risks outweigh benefits	-protect patient from falling, burns and trauma -educate that dietary intake does not correct this anemia -assess for neurologic difficulties	-educate on foods high in folic acid to consume

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	Anemia of inflammation -can be caused by cancer, autoimmunity, infection disorders (HIV, hepatitis, malaria), HF, chronic inflammation -bleeding episodes can contribute -develops 1-2 months after disease activity	-decrease of all blood cell types (pancytopenia) peripherally, and hypocellular bone marrow -most of the time due to autoimmune activity by autoreactive T lymphocytes -cytotoxic T cells target and destroy pt's own hematopoietic stem cells	-from sudden hemorrhage -trauma, surgery, conditions -can lead to hypovolemic shock	-similar to iron-deficiency anemia (bleeding ulcer, hemorrhoids, menstrual and postmenstrual blood loss)
Clinical Manifestations	-Symptoms related to the etiology (HIV, autoimmune) -chronic inflammation	-general anemia manifestations – fatigue, dyspnea, CV and cerebral responses -thrombocytopenia	-manifestations of hypovolemia (hypotension, tachycardia) -pain -internal hemorrhage -shock	-related to depletion of iron stores and is considered an irone-deficiency anemia
Diagnostic Studies	-normal cobalamin and folate levels -high serum ferritin, increased iron stores	-decreased WBC, Hgb, and platelets because all marrow is affected -High serum iron and total iron	-loss of RBC not reflected in lab data right away since blood volume loss is sudden	-CBC -same as iron-deficiency anemia

		binding capacity (TIBC) -bone marrow BX		
Drug Therapy	-blood transfusion -erythropoietin therapy -therapies for cancer	-HSCT immunosuppression with ATG and cyclosporine, steroids, and eltrombopag	-IV fluids (dextran, hetastarch, albumin, crystalloid electrolyte solutions) -PRBCs	-supplemental iron
Nursing Management	-Education on chronic diseases -therapy related to correcting underlying disorder	-identify and remove causative agent -prevent complications of infection and hemorrhage	-fluid replacement -stop blood loss -monitor drain and dressings for blood loss -give blood products -no need for long-term treatment	-identify source and stop the bleeding

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	-normal RBC's, but external factors are causing damage (autoimmune, burn, medications) -macrophages in spleen, liver, and bone marrow destroy RBC's that are old, defective and damaged	-iron overload disorder -may occur from genetics, sideroblastic anemia, liver disease, and blood transfusion	-Production and presence of increased number of RBC's. -Primary—also involves WBC's and platelets. Leads to enhanced blood viscosity, blood volume, and congestion -Secondary—Hypoxia driven or independent. Need for O2 may result from high altitude, lung disease, CVS, alveolar hypoventilation, defective o2 transport, or tissue hypoxia.
Clinical Manifestations	-general manifestations of anemia -jaundice because of increased destruction of RBC's -hepatomegaly, splenomegaly	-multiple organ failure if left untreated -early S/S: fatigue, arthralgia, impotence, ABD pain, weight loss -later s/s: hepatomegaly, cirrhosis -excess iron deposited into liver, pancreas, heart, joints, and endocrine glands and then can cause diabetes, bronzing of skin, heart problems, arthritis, and testicular atrophy	-Headache, vertigo, dizziness, tinnitus, visual changes, generalized pruritus (often from hot bath), paresthesia, erythromelalgia, angina, HG, intermittent claudication, thrombophlebitis, petechiae, bruising, nosebleeds
	-elevated bilirubin	-genetic testing -increased total iron	-Lab values seen: high hemoglobin and RBC count

Diagnostic Studies		concentration >50g -high serum iron, TIBC, serum ferritin -Liver Biopsy (amount of iron and extent of organ damage)	with microcytosis, low to normal EPO level, high WBC count with basophilia and neutrophilia, high platelet count (thrombocytosis), platelet dysfunction, high leukocyte alkaline phosphate, uric acid, cobalamin levels, high histamine -bone marrow examination
Drug Therapy	-hydrocortisone or prednisone (steroids to stop immune system from attacking RBC's)	-iron chelating agents -deferoxamine, deferasirox, deferiprone	-Myelosuppressive agents such as hydroxyurea, busulfan, chlorambucil. -Ruxolitinib inhibits expression of JAK2 mutation -low dose aspirin
Nursing Management	-maintain renal function -strict I/O's to assess for renal function	-mange diabetes, HF -education on treatment	-Reduce blood volume (300-500mL every few days) and viscosity with phlebotomy (keep hematocrit <45%)