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, or hemolysis	Group of diseases involving inadequate production of normal Hgb, which decreases RBC production; Due to an absent or reduced globulin protein.	Absence of intrinsic factor; without IF, we do not absorb cobalamin.	Can cause megaloblastic anemia; characterized by presence of abnormally large RBCs; causes impaired DNA synthesis, which results in defective RBC maturation.
Clinical Manifestations	Pallor, glossitis, cheilitis, headache, paresthesia, burning sensation of the tongue	Minor: moderate anemia w/ microcytosis and hypochromia; mild splenomegaly; bronzed skin color; bone marrow hyperplasia. Major: pale, symptoms of anemia, jaundice, pronounced splenomegaly, hepatomegaly, cardiomyopathy, chronic bone marrow hyperplasia which cause thickening of cranium and maxillary cavity; cardiac complications from iron-overload	Sore, red, beefy tongue, shiny tongue; anorexia, nausea, vomiting; weakness, paresthesia of feet and hands; reduced vibratory and position senses, ataxia, muscle weakness, and impaired cognition.	Manifestations similar to cobalamin deficiency anemia; develops insidiously. GI problems include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Thiamine deficiency which is often present with folate deficiency can cause neurologic symptoms.
Diagnostic Studies	Hgb/Hct, MCV, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin	Hbg/Hct, MCV, Reticulocytes, serum iron, TIBC, Transferrin, Ferritin, Bilirubin, Folate	RBCs appear large and have abnormal shapes. Serum cobalamin levels low. Normal serum folate levels. Serum	Low Hgb/Hct, high MCV, N or low reticulocytes, N or high serum iron, normal TIBC, slightly high

			anti-IF antibodies. Serum methylmalonic acid and homocysteine help determine cause of anemia.	transferrin, high ferritin, normal or slightly high bilirubin, N serum 12, low folate. Normal serum cobalamin level
Drug Therapy	Oral: ferrous sulfate or ferrous gluconate IM/IV: iron dextran, sodium ferrous gluconate, iron sucrose, RBC transfusion	Blood transfusion or exchange transfusions in conjunction with chelating agents that bind to iron; oral deferasirox or deferiprone, or IV/SubQ deferoxamine, luspatercept-aamt SubQ Q21 days	Cobalamin administration; parenteral vitamin B12, or intranasal cyanocobalamin. 1000mcg/day of cobalamin IM for 2 wks, then weekly until Hgb is normal, then monthly for life	1-5 mg/day PO
Nursing Management	Identify and treat underlying cause; replace iron; teach pts foods that are good sources of iron; oral or occasional IV iron supplements may be needed if diet is not enough; acute blood loss may result in packed RBC transfusion.	Drug therapy, pt may need splenectomy; monito liver, heart, lung function and provide tx as needed; Hematopoietic stem cell transplantation is the only cure.	Drug therapy as listed above. Assess for neurologic problems that are not corrected by replacement therapy; reduce risk for injury from decreased sensitivity to heat and pain; Protect pt from falling, burns, and trauma. May need physical therapy.	Replacement therapy; teach pt to eat foods high in folic acid (green leafy veggies, enriched grain products and breakfast cereals, OJ, 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. Usuaally develops 1-2 months of disease activity; causes include cancer autoimmune & infectious disorders, HF, or chronic inflammation	A disease in which a pt has peripheral blood pancytopenia (decrease of all blood cell types) and hypocellular bone marrow. Due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the pt's own hematopoietic stem cells.	Occurs with sudden bleeding; caused by trauma, surgery complications, and problems that disrupt vascular integrity	The sources of chronic blood losss are similar to those of iron-deficiency anemia (bleeding ulcer, hemorrhoids, menstrual and postmenstrual bleeding)
		Fatigue, dyspnea,	Hypotension,	Fatigue, pallor,

Clinical Manifestations	Bleeding episodes, Pallor, fatigue, SOB, headaches, dizziness, clinical signs of anemia	lethargy, increased HR and pulse pressures, pallor, headache. Susceptible to infection. Petechiae, bruising, nosebleeds	syncope, cardiac output below normal, air hunger, rapid thready pulse, cold, clammy skin, shock, lactic acidosis, death	lethargy, SOB, dizziness, headache, clinical signs of anemia
Diagnostic Studies	High serum ferritin and increased iron stores distinguish it from iron deficiency anemia. Normal folate and cobalamin blood levels	Hgb, WBCs, platelets decreased. Reticulocyte low, serum iron and TIBC may be high as initial signs of decreasing RBC production. Bone marrow biopsy, aspiration, and pathologic examination will be done to confirm laboratory findings.	Lab values don't reflect RBC loss; Values may seem normal or high for 2-3 days. Once plasma volume is replaced, RBC mass is less concentrated and RBC, Hgb, Hct levels are low and reflect actual blood loss.	Low Hgb/Hct, MCV, serum iron, TIBC, N or low bilirubin N or high reticulocytes N transferrin, ferritin, serum B12 and folate
Drug Therapy	Blood transfusions if severe; EPO therapy is used for anemia from renal disease and cancer and its therapies	Blood transfusions, HSCT; immunosuppressive therapy w/ antithymocyte globulin and cyclosporine; Eltrombopag (increase platelet counts), cyclophosphamide, alemtuzumab, androgens for those who don't respond to other tx; iron-binding agents	IV fluids, blood products, iron supplements, iron from diet	Iron supplements, IV iron transfusion, blood products if significantly low
Nursing Management	Find the underlying problem and treat it.	Remove causative agent, provide supportive therapy as pancytopenia resolves; Prevent complications from infection and bleeding.	Assess pt for pain. Replace blood volume to prevent shock, promote coagulation to prevent further bleeding, find the source of bleeding and stop the blood loss. Carefully monitor for blood loss post-op.	Assess for sources of bleeding, stop the bleeding.

Table 3	**Acquired Hemolytic**	**Hemochromatosis**	**Polycythemia**

	Anemia		
Etiology	Results form hemolysis of RBCs from extrinsic factors (physical destruction, antibody reactions, infectious agents and toxins)	Iron overload disorder characterized by increased intestinal iron absorption. Genetic defect is most common cause.	The production and presence of increased numbers of RBCs. Causes increased blood viscosity. Hypoxia driven or hypoxia independent. Chronic myeloproliferative disorder (RBCs, WBCs, platelets)
Clinical Manifestations	Pallor, fatigue, SOB, headaches, dizziness, clinical signs of anemia	Fatigue, arthralgia, impotence, abdominal pain, weight loss, liver enlargement and cirrhosis, diabetes, skin pigment changes (bronzing), heart problems (cardiomyopathy), arthritis, and testicular atrophy. Splenomegaly,	Hypertension caused by hypervolemia and hyperviscocity. Headache, vertigo, dizziness, tinnitus, visual changes, generalized itching, paresthesias and erythromelagia (painful burning and redness of feet), angina, HF, intermittent claudication, thrombophlebitis.
Diagnostic Studies	Low Hgb/Hct, N or high MCV, serum iron, ferritin, high bilirubin, high reticulocytes, N or low TIBC	High serum iron, TIBC, and serum ferritin. Testing for known genetic mutations confirms diagnosis. MRI can measure liver and cardiac iron. Liver biopsy can quantify amount of iron and establish degree of organ damage.	High Hgb/Hct, RBC mass; bone marrow examination showing hypercellularity of RBCs, WBCs, and platelets; presence of JAAK2 V617F or JAK2 exon 12 mutation. Low EPO level high WBC count with basophilia and neutrophilia, high platelet count and platelet dysfunction, normal or high leukocyte alkaline phosphatase, uric acid, and cobalamin levels.
Drug Therapy	Aggressive hydration and electrolyte replacement to reduce risk of AKI; corticosteroids, blood products, splenectomy; folate replacement, immunosuppressive agents to suppress RBC destruction, plasma exchange and eculizumab to complement protein C5.	Remove 500mL of blood each week until iron storers are depleted. Then blood is removed less often to maintain iron levels. Iron-chelating drugs may be used. Deferoxamine IV or SubQ – removes iron via kidneys; Avoid vitamin C	300-500mL of blood removed every few days until Hct reduced to acceptable levels; Reduse blood volume by 500mL every 2-3 months. Hydration therapy to reduce blood viscosity. Low-dose aspirin to prevent clotting; Myelosuppressive agents (hydroxyurea, busulfan); Ruxolitinib which inhibits expression of JAK2 mutation; alpha-Interferon-2b nad pegylated IFN aalfa-2a given to women of childbearing age or have

			intractable itching.
Nursing Management	Supportive care, initiate drug therapy, watch for signs of shock and take appropriate action, pain management.	Treatment of diabetes, cirrhosis, liver failure/cancer, HF; educate pts on avoiding vitamin C and iron supplements, uncooked seafood and iron-rich foods	Assist with or perform phlebotomy; assess intake and output during hydration therapy to avoid fluid overload; give myelosuppressive agents as ordered. Observe pt and teach them about drug side effects. Assess nutrition status; Begin activities and drug therapy to decrease thrombus formation; Assess pt for complications.

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.