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	May develop from inadequate diet intake, malabsorption, blood loss, or hemolysis. Normal iron intake is usually enough to meet the needs of men and older women (may be inadequate for people with higher iron needs i.e. menstruating women/pregnancy). Iron malabsorption may occur after certain types of GI surgery and in malabsorption syndromes, duodenal removal/diseases, blood loss. Most often blood is lost in the GI/GU systems. GI blood loss causes include peptic ulcers, gastritis, esophagitis, diverticula, hemorrhoids, and cancer. Chronic kidney disease and dialysis also contribute	A group of diseases involving inadequate production of normal Hbg, which decreases RBC production. Caused by an absent or reduced globulin protein, a-Globin chains are absent or reduced in a-thalassemia b-Globin chains are absent or reduced in b-thalassemia. Hemolysis occurs as mononuclear phagocytes in the marrow destroy most erythroblasts. Those released in the blood are rapidly destroyed by macrophages in the spleen. Commonly found in persons whose ethnic origins are near Mediterranean Sea and equatorial or near-equatorial regions of the Southeastern Asia, the Middle East, India, Pakistan, China, Southern Russia, and Africa.	Normally gastric cells secrete IF, without IF we do not absorb cobalamin. Can occur in patients who has G surgery (gastrectomy, gastric bypass) or a small bowel resection involving the ileum. Others at risk includes those with Chron disease, iletis, celiac disease, diverticula of the small intestine, or chronic atrophic gastritis. Also occurs in those with excess alcohol or hot tea ingestion, smoking, long-tern users of H2-histamine receptor blockers and proton pump inhibitors, and those who are strict vegetarians. Familiar predisposition.	Can cause megaloblastic anemia. Needed for DNA synthesis leading to RBC formation and maturation. Causes include alcohol use, diet deficiency, gastric factors such as celiac disease, gastrectomy, gastric bypass, pregnancy. Drugs interfering with absorption (metformin).
Clinical Manifestations	Early on may be asymptomatic. As it becomes chronic symptoms such as pallor, glossitis, cheilitis, fatigue, headache, parathesis, burning sensation of the tongue.	Those with thalassemia minor are often asymptomatic (mild to moderate anemia with microcytosis – small cells, and hypochromia – pale cells, mild splenomegaly, bronzed skin color,	Symptoms develop due to tissue hypoxia. GI manifestations include a sore, red, beefy, and shiny tongue, anorexia, nausea and vomiting, and abdominal pain. Neuromuscular manifestations include weakness,	Develops slowly. Symptoms may be attributed to other coexisting problems such as cirrhosis, esophageal varices. GI problems include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Similar problems to cobalamin

		and bone marrow hyperplasia). Thalassemia major is a life-threatening disease. Symptoms develop in childhood by age 2 and can cause growth and developmental defects. Person is pale, and other general symptoms of anemia, jaundice, splenomegaly, hepatomegaly, cardiomyopathy, cranium thickening and maxillary cavity.	paresthesia's of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired cognition. Develops over several months to years.	deficiency.
Diagnostic Studies	History and physical assessment, stool occult blood test, endoscopy, colonoscopy, bone marrow biopsy, CBC, serum iron, serum ferratin, serum transferrin, TIBC, Hgb, Hct, RBC, reticulocyte count	CBC, livery enzymes, Hgb, Hct, serum iron, TIBC, serum ferritin, serum transferrin, serum bilirubin.	Hgb. Hct, reticulocyte count, serum iron, TIBC, serum transferrin, serum ferratin, serum B12, RBCs. May have an upper GI endoscopy and biopsy of the gastric mucosa. Serum MMA and serum homocysteine.	Serum folate, serum cobalamin, CBC, Hgb, Hct, RBC count, reticulocyte count, serum iron, serum ferratin, serum transferrin, TIBC.
Drug Therapy	Oral iron preparations (PO, IM, IV). Parenteral iron may be given if the patient hs malabsorption, intolerance of oral iron, a need for iron beyond all oral limits, or poor patient adherence	Blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron, oral deferasiox or deferiprone, or IV or subcutaneous deferoxamine. A new therapy luspartercept-aamt may be given subcutaneously every 21 days.	Parenteral vitamin B12 (cyancobalamin, hydroxocobalamin), or intranasal cyanocobalamin.	Replacement therapy using folic acid (1-5 mg/day by mouth).
Nursing Management	Discover the underlying cause and treat, give oral/IM/IV iron, nutritional therapy, may give packed RBCs when severe.	Give medications, may require a splenectomy, monitor the liver, heart, and lung function, and provide treatment as needed. The only cure is Hematopoietic stem cell transplantation (HSCT).	Assess for neurologic problems that are not corrected by replacement therapy. Implement measures to reduce the risk for injury from the decreased sensitivity to heat and pain. Protect the patient from falls, burns, and trauma. May need physical therapy.	Educate patients to eat foods high in folic acid. Assess for similar deficiencies to cobalamin deficiencies.

Table 2	**Anemia of Chronic**	**Aplastic Anemia**	**Acute Anemia due**	**Chronic Anemia**

	Disease		to Blood Loss	due to Blood Loss
Etiology	(Anemia of inflammation) usually develops after 1 to 2 months of disease activity. Causes include cancer, autoimmune and infectious disorders, HF, or chronic inflammation. Bleeding episodes can contribute to anemia of chronic disease. Associated with an underproduction of RBCs and mild shortening of RBC survival. RBCs are normally normocytic, normochromic, and hyperproliferative. The anemia is normally mild and can become more severe if the underlying disorder is not treated.	About 70% of aplastic anemias are due to autoimmunity activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells. Other causes include toxic injury to bone marrow stem cells or an inherited stem cell effect. Other causes include chemical agents and toxins, drugs (NSAIDs), immunosuppression, radiation, toxic injury, viral/bacterial infection.	Occurs with sudden bleeding. Causes include trauma, surgery complications, and problems that disrupt vascular integrity. Two clinical concerns (first: sudden reduction in the total blood volume which can lead to hypovolemic shock, second: if the acute loss is more gradual, the body maintains is volume by slowly increasing the plasma volume, RBCs are decreased).	Sources of blood loss are similar to those of iron deficiency anemia (hemorrhoids, menstrual blood loss). Effects are usually due to depleted iron stores.
Clinical Manifestations	Tiredness, weakness, SOB, pallor, yellow skin, irregular heartbeat, dizziness, CP, cold hands/feet	Can manifest abruptly (over days) or insidiously (over weeks/months). Can me mild to severe. The patient may have symptoms caused by suppression of any or all bone marrow elements. General manifestations of anemia (fatigue, dyspnea) as well as cardiovascular and cerebral responses. Low neutrophils may lead to infection (risk of septic shock/hemorrhage).	Caused by the body's attempts to maintain an adequate blood volume and meet O2 consumptions. Symptoms depend on blood loss. 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 CO below normal at rest, air hunger, rapid,	Blue color to the whites of the eyes, brittle nails, desire to eat ice, lightheadedness, pallor, SOB with activity or at rest.

			thready pulse, cold, clammy skin) 50%/2500 mL (shock, lactic acidosis, potential death)	
Diagnostic Studies	Serum ferritin and serum iron, CBC, RBC, Hgb, Hct, TIBC.	Hgb, WBC, platlet values, RBC, reticulocyte count, serum iron, TIBC, bone marrow biopsy.	CBC, test plasma, RBC, Hgb, Hct, platlet counts, INR, PTT, urine sample	CBC, RBC count, Hgb, Hct, serum iron, serum ferratin, serum transferrin, TIBC, stool examination for occult blood.
Drug Therapy	Correct the underlying problem. If the anemia is severe, blood transfusion may be needed (not recommended for long-term treatment) EPO therapy is used for anemia from renal disease and cancer and its therapies.	Immunosuppressive therapy with antithymocyte globulin (ATG) and cyclosporin. Eltrombopag can increase platelet counts. High dose cyclophosphamide, alemtuzumab, or androgens may be helpful. Blood transfusions if needed.	Replace blood volume using PRBC, if a large amount of volume is lost give whole blood, platelets, plasma, and cryoprecipitate may be given. IV fluids given include 0.9% NaCl, LR. May need iron supplements.	Iron supplements.
Nursing Management	Treat the underlying cause and use medications such as packed RBCs when needed.	Identify and treat the underlying cause. Consider HSCT.	Assess for pain, monitor post-op patients and monitor their blood loss from their dressings, tubes, etc. Anemia should be able to correct itself. Find the source of bleeding, control the blood loss, and replace fluid and blood volumes. No long term treatments.	Identify the cause and stop the bleeding. Do a history and physical assessment

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

	Anemia		
Etiology	Results from hemolysis of RBCs from extrinsic factors (physical destruction, antibody reactions, infectious agents, and toxins). Physical destruction of RBCs results from the exertion of extreme force on the cells. Traumatic events that disrupt the RBC membrane include hemodialysis, extracorporeal circulation used in cardiopulmonary bypass, and prostatic heart valves (may damage RBCs). RBCs may be fragmented and destroyed as they try to pass though abnormal arterial or venous microcirculation. RBCs are sheared as they try to pass by excess platelet aggregation and/or fibrin polymer formation, such as seen in thrombotic thrombocytopenic purpura (TTP) and disseminated intravascular coagulation (DIC). People develop antibodies to their own RBCs, and this can be idiopathic in cause or autoimmune, or from medications. Infectious agents cause hemolysis in three ways (invading the RNC and destroying its contents, releasing hemolytic substances, generating an antigen-antibody reaction). Various agents may be toxic to RBCs and cause hemolysis (chemicals).	Iron overload disorder characterized by increased intestinal iron absorption. Genetic defect is the most common cause. May occur with diseases such as sideroblastic anemia and liver disease, and the chronic blood transfusions used to treat thalassemia and SCD. Exceed normal iron counts in the blood.	Production and presence of increased numbers of RBCs. The increase in RBCs can be so great that blood circulation is impaired because of the increased blood viscosity and volume. There are two types Primary polycythemia: chronic myeloproliferative disorder (involves increased production not only of RNCs but also WBCs and platelets). Leads to enhanced blood viscosity and blood volume and congestion of organs and tissues with blood. Splenomegaly and hepatomegaly are common. Predisposed to clotting. Chronic course. Secondary polycythemia: can either be hypoxia driven or hypoxia independent. In hypoxia-driven, hypoxia stimulates the kidneys to make EPO, which stimulates RBC production, need for O2 may be due to high altitude, etc. EPO levels may return to normal once Hgb stabilizes at a higher level. Physiologic response in which the body tries to compensate for a problem. In hypoxia-independent, cancer or benign tumor tissue makes EPO.
Clinical Manifestations	Hemolytic crisis is a risk. Weakness, pallor, jaundice, dark-colored urine, fever, inability to do physical activity, and heart murmur.	Symptoms usually do not develop until after age 40 in men and after 50 in women. Early symptoms are nonspecific (fatigue, arthralgia, impotence, abdominal pain, and weight loss). Later, excess iron accumulates in the liver and causes liver enlargement and cirrhosis. Excess iron deposits in the liver, pancreas, heart, joints, and endocrine glands	Circulatory manifestations occur due to the HTN caused by hypervolemia and hyperviscosity. Symptoms include headache, vertigo, dizziness, tinnitus, and visual changes. Generalized itching (exacerbated by a hot bath) may be a striking symptom – related to histamine release from increased number of basophils. Parathesis and

		cause diabetes, skin pigment changes (bronzing), heart problems, arthritis, and testicular atrophy. May have enlarged liver and spleen and skin pigmentation changes.	erythromelalgia (painful burning and redness of the hands and feet). Patient may have angina, HF, intermittent claudication, and thrombophlebitis, which may be complicated by embolization. Manifestations caused by blood vessel distention, impaired blood flow, circulatory stasis, thrombosis, and tissue hypoxia from the hypervolemia and hyperviscosity. May have petechiae, bruising, nosebleeds, or GI bleeding (may be acute or catastrophic). Hepatomegaly and splenomegaly may cause satiety and fullness. May have peptic ulcers, plethora.
Diagnostic Studies	Hgb, Hct, RBC count, reticulocyte count, serum iron, serum ferratin, serum transferrin, TIBC, stool exam for occult blood.	Serum iron, TIBC, and serum ferratin, MRI, liver biopsy, CBC.	Hgb, Hct, RNC, bone marrow examinations, WBCs, platelets, EPO level.
Drug Therapy	Corticosteroids, fluid/electrolyte replacement. Folate replacement. Immunosuppressive agents (glucorticoids or rituximab).	Iron-chelating drugs may be used. They form a complex with iron and promote its excretion from the body. Deferoxamine chelates and removed iron via the kidneys (given IV or subcutaneously). Deferasirox and deferiprone are PO drugs.	Myelosuppressive agents, such as hydroxyurea, or busulfan may be given (ruxolitinib).
Nursing Management	General supportive cause until causative agent is found and eliminated. Be ready for emergency therapy (hemolytic crisis) – aggressive hydration, electrolyte replacement.	Remove excess iron form the body and minimize any symptoms the patient may have. Iron removal is achieved by removing 500 mL of blood each week until the iron stores are depleted. Then removing the blood less often from then on. Diet changes should be educated on including avoiding vitamin C and iron supplements, uncooked seafood, and iron-rich foods. Manage organ involvement with the usual treatments for the problems.	Reduce blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment (reduce Hct). Depends on agency, may assist with phlebotomy. Assess I&O, give drugs, observe patient, and educate. Assess patient's nutritional status. Requires ongoing evaluation. Assess 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.