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	Develop from inadequate diet intake, malabsorption, blood loss, or hemolysis	Inadequate production of normal Hgb, which decreases RBC production, it is due to an absent or reduced globulin protein	No secretion of intrinsic factor(IF), which is required for cobalamin (extrinsic factor) absorption	Needed for DNA synthesis leading to RBC formation and maturation
Clinical Manifestations	Early on the pt may or may not have symptoms. As it becomes chronic, any general manifestations of anemia occur, such as pallor (most common), glossitis (inflammation of tongue) is the second most common, cheilitis (inflammation of lips), headache, paresthesias, and burning sensation of the tongue.	Often asymptomatic, they have mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Thalassemia major is a self-threatening, starts at age 2 and causes growth and development deficits: pale, jaundice from RBC hemolysis, pronounced splenomegaly, hepatomegaly, cardiomyopathy, thickening of the cranium and maxillary cavity	GI manifestations: sore, red, beefy, and shiny tongue; anorexia, N/V, and abdominal pain. Neuromuscular manifestations: weakness, paresthesias of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired cognition. It may take several months or years for manifestations to develop.	Develops insidiously. Symptoms may be attributed to other coexisting problems (cirrhosis, esophageal varices). GI problems: stomatitis, cheilosis, dysphagia, flatulence, and diarrhea
Diagnostic Studies	Hgb and Hct (decreased), MCV (decreased), reticulocytes (N or slight Increase or	Hgb and Hct (decreased), MCV (N or decreased), Reticulocytes	Hgb and Hct (decreased), MCV (increased), Reticulocytes (N or	Hgb and Hct (decreased), MCV (increased), Reticulocytes (N or

	decrease), serum iron (decrease), TIBC (increase), Transferrin (N or decrease), Ferritin (decrease), bilirubin (N or decrease), Serum B12 (N), Floate (N), stool occult blood test, endoscopy and colonoscopy, bone marrow biopsy	(increased), Serum iron (increased), TIBC (decreased), Transferrin (decreased), Ferritin (N or increased), Bilirubin (increased), Serum B12 (N), Folate (decreased), RBC, O2, lung disease, HTN, diabetes, growth retardation, and hypogonadiam	decreased), Serum iron (N or increased), TIBC (N), Transferrin (slight increase), Ferritin (increased), Bilirubin (N or slight increased), Serum B12 (decreased), Folate (N), RBCs appear large (macrocytic) and have abnormal shapes, serum cobalamin levels are low, serum test for anti-IF antibodies, upper GI endoscopy and biopsy, MMA and serum homocysteine help determine the cause of anemia.	decreased), Serum iron (N or increased), TIBC (N), Transferrin (slight increase), Ferritin (increased), Bilirubin (N or slight increased), Serum B12 (N), Folate (decreased)
Drug Therapy	1. Iron best absorbed in the duodenum and proximal jejunum 2. daily dose should provide 100-200mg of elemental iron, taken 3-4x a day each capsule has 60-70mg of iron 3. Iron best absorbed in acidic environment, take without food an hour before meals 4.undiluted iron can stain teeth, so dilute and take through a straw 5. GI side effects may occur, so sit upright for 30min after Vitamin C helps with the absorption of iron Will cause stools to be black and constipation can occur.	Does not need treatment because the body adapts to the reduction of normal Hgb. Treatment of thalassemia major includes blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron. Drugs used include oral deferasirox or deferiprone, or IV or subcutaneous deferoxamine. A new therapy luspatercept-aamt may be given subcutaneously q21days it improves Hgb levels and reduces transfusion needs. Hematopoietic stem cell transplantation (HSCT) is the only	Parenteral vitamin B12 or intranasal cyanocobalamin. 1000mcg/day of cobalamin IM for 2 weeks, then weekly until the Hgb is normal, and then monthly for life	Replacement therapy, the usual dosage is 1-5 mg/day PO. The duration of the treatment depends on the reason for the deficiency.

		cure for thalassemia, with iron chelation therapy pts are living longer		
Nursing Management	Treat the underlying problem causing the iron loss, reduced intake (malnutrition, alcohol use), or poor iron absorption, iron replacement. Teach foods that are good sources of iron, if diet good, then they may need to oral or occasional IV iron supplements. If iron deficiency is from acute blood loss, pt may need packed RBC transfusion.	A splenectomy may be needed, so monitor liver, heart, and lung function and provide treatment as needed. Check Hgb levels and iron levels and give transfusions when needed	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 pt from falls, burns, and trauma. Some may need physical therapy	Teach the pt to eat foods high in folic acid (green leafy vegetables, enriched grain products and breakfast cereals, orange juice, peanuts and avocados.

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Underproduction of RBCs and mild shortening of RBC survival.	Autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells. Incidences are rare, 2 new cases per million ppl/year	Trauma, surgery complications, and problems that disrupt vascular integrity. A sudden reduction in the total blood volume can lead to hypovolemic shock or if the acute loss is more gradual, the body maintains its blood volume by slowly increasing the plasma volume. The number of RBC carrying O2 is decreased.	Bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss. The effects of chronic blood loss are usually due to depleted iron stores. Hemolysis anemia is caused by the destruction or hemolysis of RBCs at a rate that exceeds production.
	Must be	Manifest abruptly	Postural	Jaundice occurs due

Clinical Manifestations	recognized from anemia of other causes. High serum ferritin and increased iron stores distinguish it from iron deficiency anemia. It usually develops 1-2 months of disease activity.	over days or insidiously over weeks to months. Vary from mild to severe. Suppression of any and all bone marrow elements. Fatigue, dyspnea, cardiovascular and cerebral responses may occur. Pt w/ neutropenia is susceptible to infection, they are at risk for septic shock or death. Thrombocytopenia can lead to bleeding (petechiae, bruising, nosebleeds)	hypotension, pain (internal bleeding can cause pain d/t tissue distention, organ displacement, and nerve compression. Numbness and pain in the lower extremities from compression of the lateral cutaneous nerve, change in BP, decrease in CO, thready pulse, cool clammy skin, the major complication is shock	to the increased destruction of RBCs causing increased bilirubin levels. Enlarged spleen and liver from hyperactivity, which is due to macrophage phagocytosis of the defective RBCs, increased HR, blurred vision, anorexia, sensitivity to cold, wt loss, lethargy, tachypnea, pallor, itching, headache, vertigo, impaired thought process
Diagnostic Studies	Cytokines, interleukin-6, iron, RBC levels, the life span of the RBCs, decreased erythropoietin (EPO) production, and an ineffective bone marrow response to EPO. High serum ferritin and increased iron stores	Hgb and Hct (decreased), MCV (N or slight increase), Reticulocytes (decreased), Serum iron (N or increased), TIBC (N or increased), Transferrin (N), Ferritin (N), Bilirubin (N), Serum B12 (N), Folate (N), RBC normal, bone marrow biopsy, aspiration, and pathologic examination to confirm lab findings. Getting a HSCT done	Plasma volume has not had time to increase from sudden blood loss, lab data does not reflect RBC loss. Values seem normal or high for 2-3 days. Once the plasma volume is replaced, the RBC mass is less concentrated, then, RBC, Hgb, Hct levels are low and reflect the actual blood loss	Hgb and Hct (decreased), MCV (decreased), Reticulocytes (N or increased), Serum iron (decreased), TIBC (decreased), Transferrin (N), Ferritin (N), Bilirubin (N or decreased), Serum B12 (N), Folate (N), RBC levels, BUN and creatinine, ultrasound, LFT
Drug Therapy	Correct the underlying problem. If severe, blood transfusions may be needed, they are not recommended for long-term treatment. EPO therapy is used for anemia from renal disease and cancer and its therapies	Managed with EPO or blood transfusions. Identifying and removing the causative agent and providing supportive care until the pancytopenia resolves. Given blood transfusions, immunosuppressive	Replace the blood volume to prevent shock, promote coagulation to prevent further bleeding. Blood transfusions (packed RBCs), if a large amount of blood loss, whole blood, platelets, plasma, and cryoprecipitate	Iron supplements, packed RBCs replacement, IV fluids, promote coagulation to prevent bleeding further, Whole blood transfusion, platelets and plasma transfusions

		therapy with ATG and cyclophosphamide. Eltrombopag increases platelet counts, iron-binding agents are used to prevent iron overload.	may be given because large volumes of RBCs dilute the coagulation. IV fluids used in emergencies (0.9% NaCl, lactated Ringers) or colloids that pull fluid in vascular space (dextran, hetastarch, albumin) iron supplements	
Nursing Management	Blood transfusions, drug therapy, and O2 therapy to stabilize the pt, diet and lifestyle changes can help reverse it, encourage rest and activity periods, minimize fatigue, monitor cardiorespiratory response to activity and collaborate with the dietitian to determine the number of calories and types of nutrients that the pt may need	Aimed at preventing complications from infection and bleeding.	Replace the blood volume to prevent shock, promote coagulation to prevent further bleeding, and finding the source of the bleed and stopping it. Give iron supplements or diet changes, if adequate enough. Carefully monitor the blood loss from various drainage tubes and dressings and implement appropriate actions postoperatively. Replace the fluids that are lost, there should be no long-term treatment for this anemia	Identify the source and stop the bleeding. The pt may need iron supplements. Maintain renal function and checking accurate intake and output for the function of the kidneys. O2 therapy may be needed to supply vital functions. Monitor cardiorespiratory response to activity and collaborate with the dietitian to determine the number of calories and types of nutrients that the pt may need

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	RBCs are normal but external factors are causing damage. Macrophages, particularly those in the spleen, liver, and bone marrow, destroy RBCs that are old, defective, or moderately damaged.	Iron overload disorder characterized by increased intestinal iron absorption. A genetic defect is the most common cause. It may occur with diseases such as sideroblastic anemia and liver disease, and the chronic blood transfusions used to treat thalassemia and SCD	Increased numbers of RBCs. The increase of RBCs can be so great that blood circulation is impaired d/t increased blood viscosity and volume. 2 types of polycythemia, are primary polycythemia or polycythemia vera, and

			secondary polycythemia. Primary is a chronic myeloproliferative disorder, increased production of RBCs, WBCs, and platelets. Secondary can either be hypoxia driven or hypoxia independent.
Clinical Manifestations	Jaundice occurs due to the increased destruction of RBCs causing increased bilirubin levels. Enlarged spleen and liver from hyperactivity, which is due to macrophage phagocytosis of the defective RBCs, increased HR, blurred vision, anorexia, sensitivity to cold, wt loss, lethargy, tachypnea, pallor, itching, headache, vertigo, impaired thought process, glossitis, smooth tongue, icteric conjunctiva and sclera	Do not develop until after age 40 years in men and after 50 years in women. Early symptoms are nonspecific. Fatigue, arthralgia, impotence, abdominal pain, and weight loss. Later, the excess iron accumulates in the liver and causes liver enlargement and cirrhosis, excess iron deposits in the liver, pancreas, heart, joints, and endocrine glands cause diabetes, skin pigment changes (bronzing), heart problems (cardiomyopathy), arthritis, and testicular atrophy. There may be an enlarged liver and spleen and skin pigmentation changes.	Circulatory manifestations are the fort signs: headache, vertigo, dizziness, tinnitus, and visual changes. Generalized itching (exacerbated by hot bath), paresthesias and erythromelalgia (painful burning and redness of the hands and feet), angina, HF, intermittent claudication, thrombophlebitis. Blood vessel distention, impaired blood flow, circulatory stasis, thrombosis, and tissue hypoxia. The highest mortality is stroke from the thrombus. Petechiae, bruising, nosebleeds, or GI bleeds, hepatomegaly, splenomegaly, pian from peptic ulcers, plethora (ruddy complexion)
Diagnostic Studies	Hgb and Hct (decreased), MCV (decreased), Reticulocytes (N or increased), Serum iron (decreased), TIBC (decreased), Transferrin (N), Ferritin (N), Bilirubin (N or decreased), Serum B12 (N), Folate (N), RBC levels, BUN and creatinine, ultrasound, LFT	High serum iron, TIBC, and serum ferritin. Testing for known genetic mutations confirms the diagnosis. MRI can measure liver and cardiac iron, liver biopsy can quantify the amount of iron and establish the degree of organ damage.	Primary: high Hgb, Hct, and RBC mass; bone marrow examination showing hypercellularity of RBCs, WBCs, and platelets; presence of JAK2 or V617F or JAK2 exon 12 mutation. Other studies show low EPO level (secondary polycythemia has a high level), high WBC count with basophilia and neutrophilia, high platelet count and platelet dysfunction, and normal or high leukocyte alkaline phosphate, uric acid, and cobalamin levels.
Drug Therapy	Iron supplements, packed RBCs replacement, IV	Iron-chelating drugs can be used, they form complex with iron and promote its	Low dose aspirin to prevent clotting. Myelosuppressive agents, such as

	fluids, promote coagulation to prevent bleeding further, Whole blood transfusion, platelets and plasma transfusions	excretion from the body. Deferoxamine chelates and removes iron via the kidneys. Deferasirox and deferiprone are oral drugs. Avoid vitamin C and iron supplements.	hydroxyurea, or busulfan, ruxolitinib, which inhibits expression of JAK2 mutation and is given to those who do not respond to hydroxurea. Alpha-interferon-2b and pegylated IFN alfa-2a are options for women of childbearing age or those with intractable itching.
Nursing Management	Identify the source and stop the bleeding. The pt may need iron supplements. Maintain renal function and checking accurate intake and output for the function of the kidneys. O2 therapy may be needed to supply vital functions. Monitor cardiorespiratory response to activity. Monitor Hgb levels to prevent obstruction of the renal tubules.	Goal is remove excess iron from the body and minimize any symptoms the pt may have. Iron removal is achieved by removing 500mL of blood each week until the iron stores are depleted. Then blood is removed less often to maintain iron lead levels within normal limits. Diet changes include avoiding vitamin C and iron supplements, uncooked seafood, and iron-rich foods. Manage problems from organ involvement (diabetes, HF) with the usual treatment of these problems. Most common causes of death are cirrhosis, liver failure, liver cancer, and HF. Early diagnosis life expectancy is normal.	Assist with or perform the phlebotomy, assess intake and output during hydration therapy to avoid fluid overload or fluid deficit. Give myelosuppressive agents as ordered, Observe the pt and teach them about drug side effects. Assess nutrition status, inadequate intake can result in GI symptoms of fullness, pain, and dyspepsia. Begin activity and drug therapy to decrease thrombus. Requires ongoing evaluation and assess pt 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.