Elizabeth McCloy

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	Based on clinical condition causing the anemia. May develop from inadequate diet intake, malabsorption, blood loss, or hemodialysis.	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. Normally, the parietal cells of the gastric mucosa secrete IF. IF is required for cobalamin (extrinsic factor) absorption.	Can cause megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	Result from the body's response to hypoxia Mild anemia: Hbg 10-12 g/dL underlying disease or has a compensatory response to heavy exercise. Palpitations, dyspnea, and mild fatigue. Moderate anemia: Hbg 6-10 g/dL Increase in cardiopulmonary symptoms (sym. while resting or activity). Severe anemia: Hbg less than 6 g/dL symptoms involving multiple body symptoms. Cardio:↑HR, ↑pulse pressure, sys murmurs,	Minor is often asymptomatic. May have mild to moderate anemia with microcytosis and hypochromia, mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Symptoms develop in childhood by age 2 and can cause growth and development deficits. Pale and has other symptoms.	General manifestations of anemia d/t cobalamin deficiency develop d/t tissue hypoxia. GI manifestations: sore, red, beefy, and shiny tongue; anorexia, N/V, and abd pain. Typical neuromuscular manifestations: weakness, paresthesia of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired cognition.	Similar to cobalamin deficiency. It develops insidiously. Symptoms may be attributed to other coexisting problems. GI problems may include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Thiamine deficiency, which is often present with folate deficiency, causes neurologic symptoms.

	inter claudication, angina, HF, MI. Eyes: Icteric conjunctiva, and sclera, retinal hemorrhage, blurred vision. GI: anorexia, hepatomegaly, splenomegaly, problems swallowing, sore mouth. General: sensitivity to cold, weight loss, lethargy. Mouth: glossitis, smooth tongue. Musculoskeletal: Bone pain. Pulmonary: tachypnea, orthopnea, dyspnea at rest. Neurologic: headache, vertigo, irritability, depression, impaired thought processes. Skin: pallor, jaundice, itching. Glossitis is the second most common.		May take several months or years for manifestations to develop.	
Diagnostic Studies	Stool occult blood test: iron deficiency. Endoscopy and colonoscopy may detect GI bleeding. Bone marrow biopsy may be done if other tests are inconclusive. H&P, hgb and Hct levels, RBC count, including morphology, Reticulocyte count, serum iron, serum transferrin, total iron-binding capacity.	Genetic testing CBC count and peripheral blood smear, iron studies, skeletal survey, ECG, HLA typing.	Hgb/Hct: ↓ MCV: ↑ Reticulocytes: N or ↓ Serum iron: N or ↑ TIBC: N Transferrin: Slight ↑ Ferritin: ↑ Bilirubin: N or slight ↑ Serum $B_{12:}$ ↓	Hgb/Hct: ↓ MCV: ↑ Reticulocytes: N or ↓ Serum iron: N or ↑ TIBC: N Transferrin: Slight ↑ Ferritin: ↑ Bilirubin: N or slight ↑ Serum $B_{12:}$ ↓

			Folate: N	Folate: N Serum folate level is low (normal is 5-25 ng/mL [11 to 57 nmol/L], with a normal serum cobalamin level.
Drug Therapy	Iron 100-200mg Oral: ferrous sulfate or ferrous gluconate IM or IV: iron dextran, sodium ferrous gluconate, iron sucrose	Oral deferasirox or deferiprone, or Ivor subcutaneous deferoxamine. Luspatercept-aamt may be given subcut every 21 days	Cobalamin administration: without this the patient will die in 1-3 years Parental vitamin B_{12} or intranasal cyanocobalamin 1000 mcg/day of cobalamin IM for 2 weeks, then weekly until the Hgb is normal, and then monthly for life.	Replacement therapy. Usual dosage is 1-5 mg/day by mouth.
Nursing Management	Identify and treat the underlying cause, packed RBC transfusion.	Major: assess for severe anemia, splenomegaly, or hepatomegaly, with abdominal enlargement, frequent infections, bleeding tendencies (epistaxis, and anorexia) Intermediate: Assess for anemia, jaundice, and splenomegaly, hemosiderosis caused by increased intestinal absorption of iron. Minor: Assess for mild anemia usually	Assess for neurological 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 falling, burns, and trauma. May need physical therapy.	Correcting the cause of the anemia Ex. Blood transfusions, drug therapy and O_2 therapy to stabilize pt. For the pt with fatigue, encourage alt. rest and activity periods.

		with no s/s.		

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Usually develops after 1 to 2 months of disease activity.	About 70% of aplastic anemias are d/t autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the pts own	Occurs with sudden bleeding. Causes of acute blood loss include trauma, surgery complications, and problems that disrupt vascular integrity. Clinical	Based on clinical condition causing the anemia. May develop from inadequate diet intake, malabsorption, blood loss, or hemodialysis.

		hematopoietic stem cells. Other causes include toxic injury to bone marrow stem cells or an inherited stem cell defect.	concerns: a sudden reduction in the total blood volume can lead to hypovolemic shock. If the acute loss is more gradual, the body maintains its blood volume by slowly increasing the plasma volume.	
Clinical Manifestations	Cancer, autoimmune and infectious disorders (HIV, hepatitis, malaria), HF, or chronic inflammation. Bleeding episodes can contribute to anemia.	Can manifest abruptly (over days) or insidiously over weeks to months. It can vary from mild to severe. Pt will have symptoms caused by suppression of any of all bone marrow elements. General manifestations of anemia, such as dyspnea, fatigue, cardiovascular and cerebral responses may occur. Pt with neutropenia (low neutrophil count) is susceptible to infection. At risk for septic shock and death. Thrombocytopenia can lead to bleeding.	Caused by the body's attempts to maintain an adequate blood volume and meet O2 requirements. Internal bleeding may cause pain d/t tissue distention, organ displacement, and nerve compression. Pain may be localized or referred. Major complication is shock.	Usually d/t depleted iron stores.
Diagnostic Studies	Underproduction of RBCs and mild shortening of RBC survival.High serum ferritin and increased iron stores distinguish it from iron deficiency anemia. Normal folate and cobalamin blood	Lab studies confirm the dx. Hgb, WBC, and platelet values are decreased. Normocytic, normochromic anemia. Reticulocyte count is low. Serum iron	Values may seem normal or high for 2-3 days. However once the plasma volume is replaced, the RBC mass is less concentrated. Then, RBC, Hgb, and Hct levels are low to	Considered an iron deficiency anemia. Based on clinical condition causing the anemia. May develop from inadequate diet intake, malabsorption, blood

	levels. Gastritis, hemorrhoids, menstrual flow	and total iron-binding capacity may be high as initial signs of decreased RBC production. Bone marrow biopsy, aspiration, and pathologic examination will be done to confirm the laboratory findings. The marrow in aplastic anemia is hypocellular with increased yellow marrow (fat content).	reflect the actual blood loss.	loss, or hemodialysis.
Drug Therapy	Best tx is to correct the underlying problem. If the anemia is severe, blood transfusions may be needed. EPO therapy is used for anemia from renal disease but is limited d/t increased risk for thromboembolism and death in some pts.	Early consideration of an HSCT is critical. The best treatment outcomes for those who receive and HSCT occur in younger patients who have had limited blood transfusions. Prior transfusions increase the risk for HSCT graft rejection. Those in interim or ineligible for HSCT may receive immunosuppressive therapy with antithymocyte globulin and cyclosporine. Eltrombopag, and oral thrombopoietin receptor agonist, can increase platelet counts/	Blood transfusions (packed RBCs) can be used depending on the volume lost. If a large volume of blood is lost, whole blood, platelets, plasma, and cryoprecipitate may be given bc large volume of RBCs dilute the coag system. IV fluids used in emergencies include volume replacement. May need iron supplements	Management of chronic blood loss anemia involves identifying the source and stopping the bleeding. May need iron supplements.

Nursing Management	Encourage alternate rest and activity periods. Monitor the patient's cardiorespiratory response to activity (tachycardia, dysrhythmias, dyspnea, pallor, tachypnea). Collaborate with the dietitian to determine the number of calories and type of nutrients needed to meet nutrition requirements.	Identify and remove the causative agent (when possible) and providing supportive care until the pancytopenia resolves. Encourage alternate rest and activity periods. Monitor the patient's cardiorespiratory response to activity (tachycardia, dysrhythmias, dyspnea, pallor, tachypnea). Collaborate with the dietitian to determine the number of calories and type of nutrients needed to meet nutrition requirements.	Assess the patient for pain. Replacing blood volume to prevent shock, promoting coagulation to prevent further bleeding, and finding the source of bleeding and stopping it.	Identify and treat the underlying cause, packed RBC transfusion. Encourage alternate rest and activity periods. Monitor the patient's cardiorespiratory response to activity (tachycardia, dysrhythmias, dyspnea, pallor, tachypnea). Collaborate with the dietitian to determine the number of calories and type of nutrients needed to meet nutrition requirements.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Results from hemolysis of RBCs from extrinsic factors. Factors include physical destruction, antibody reactions, and infectious agents and toxins	An iron overload disorder characterized by increased intestinal iron absorption. A genetic defect is the most common cause.	Two types of polycythemia, primary polycythemia, or polycythemia vera and secondary polycythemia. Their causes and pathogenesis differ, although their complications and manifestations are similar.
Clinical Manifestations	Impaired platelet production: Cancer and other disorders (aplastic anemia, leukemia, lymphoma, myeloma, myelodysplastic disorders), marrow metastases by solid tumors. Drugs: chemotherapy, others. Immune thrombocytopenia. Infections, bacterial, fungal, viral. Nutrition deficiencies, alcohol use. radiation. Increased platelet destruction: artificial surfaces, DIC, HIT, pregnancy related. Thrombotic microangiopathy, atypical hemolytic uremic syndromes, Thrombotic thrombocytopenic purpura. Abnormal platelet	Symptoms usually do not develop until after age 40 years in men and after 50 years in women. Early symptoms are nonspecific including fatigue, arthralgia, impotence, abd pain, and weight loss. Later, the excess iron accumulates in the liver, pancreas, heart, joints, and endocrine glands cause diabetes, skin pigment changes, heart problems (cardiomyopathy), arthritis, and testicular atrophy.	Circulatory manifestations occur d/t hypertension caused by hypervolemia and hyperviscosity. First manifestations include headache, vertigo, dizziness, tinnitus, and visual changes. Generalized itching may be a striking symptom. Pt may have angina, HF, intermittent claudication, and thrombophlebitis.

	distribution: dilution (massive blood transfusion, fluids), splenic sequestration.		
Diagnostic Studies	Stool occult blood test: iron deficiency. Endoscopy and colonoscopy may detect GI bleeding. Bone marrow biopsy may be done if other tests are inconclusive. H&P, hgb and Hct levels, RBC count, including morphology, Reticulocyte count, serum iron, serum transferrin, total iron-binding capacity.	Lab values show a high serum iron, TIBC, and serum ferritin. Testing for known genetic mutations confirms the dx. MRI can measure liver and cardiac iron. Liver biopsy can quantify the amount of iron and establish the degree of organ damage. With early dx and tx, life expectancy is normal.	Phlebotomy is the mainstay of treatment. Major diagnostic criteria for polycythemia vera include high Hgb, HCT, and RBC mass, and bone marrow examination showing hypercellularity of RBCs, WBCs, and platelets, and presence of JAK2 V617F or JAK2 exon 12 mutation
Drug Therapy	May need folate replacement. To suppress the RBC destruction, immunosuppressive agents may be used, such as glucocorticoids, or rituximab. For severe cases, hemolysis, thrombocytopenia and acute kidney injury additional	Goal of treatment is to remove excess iron from the body and minimize symptoms the pt may have. Iron removal is achieved by removing 500 mL of blood each week until the iron stores are depleted. Iron-chelating drugs may be used. Deferoxamine chelates and removes iron via the	myelosuppressive agents, hydroxyurea or busulfan may be given. Ruxolitinib inhibits expression of the JAK2 mutation, given to those who don't respond to hydroxyurea. Low dose aspirin to prevent

	immunosuppressants may be used. Supportive care may include giving corticosteroids and blood products or removing the spleen.	kidneys: given IV or subcut. Deferasirox and deferiprone are oral drugs. Diet changes include avoiding Vit C and iron supplements, uncooked seafood, and iron-rich foods.	clotting.
Nursing Management	General supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs. This includes aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by Hgb clogging the kidney tubules and subsequent shock. Supportive care may include giving corticosteroids and blood products or removing the spleen.	Teach dietary needs. Manage problems from organ involvement with the usual tx for these problems. The most common causes of death are cirrhosis, liver, failure, liver cancer, and HF.	Assess the pts nutrition status. Inadequate food intake can result from GI symptoms of fullness, pain and dyspepsia. Assess the patient for complications with each encounter. May assist with or perform phlebotomy Assess Is & Os during hydration therapy to avoid deficit Give myelosuppressive agents as ordered. Observe the patient and teach them about drug side effects.

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.

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