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	Decreased RBC production decreases hemoglobin synthesis.	Disease that involved inadequate production of normal Hemoglobin, which decreases RBC production. Thalassemia is caused by the absences or reduction of globin proteins. Thalassemia has an autosomal recessive genetic gene.	Caused by the absence of intrinsic factor (IF). Normally begins usually after age 40-60 years of age.Cobalamin is normally absorbed in the distal ileum. Without IF, cobalamin will not be absorbed.	Folic acid (folate) deficiency also causes megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	Mild: • Palpitations • fatigue Moderate: • Increased palpitations (Bounding pulse) • SOB Severe: • Tachycardia • Increase pulse pressure	Thalassemia minor is often asymptomatic. Mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells) experience mild splenomegaly, bronzed skin, and bone marrow hyperplasia.	• Tissue hypoxia • GI manifestations such as sore, red, beefy and shiny tongue. N/V and abdominal pain. • Weakness • Ataxia • Dementia	The patient's symptoms may be attributed to other coexisting problems (Ex: cirrhosis, esophageal varices) GI problems may include Stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Thiamine deficiency can also occur and may also cause neurologic symptoms.

	• angina • HF • Blurred vision • Wt. loss • Sensitivity to cold • Pallor, Jaundice and pruritus • Tachypnea • vertigo • headache • Impaired thought • SOB at rest	Thalassemia Major is a life-threatening disease in which growth both physical and mental is often slowed. Normally a pale and displays other general symptoms of anemia.		
Diagnostic Studies	Blood test: Look at hemoglobin levels	Blood tests can reveal the number of red blood cells and abnormalities in size, shape or color. Blood tests can also be used for DNA analysis to look for mutated genes.	Blood test: looking at RBC, Folate levels (low),anti-IF antibodies, serum methylmalonic acid (MMA) and serum homocysteine (High in both). An Upper GI endoscopy and biopsy of the gastric mucosa can give a diagnosis.	Blood test to see folic acid deficiency (normal 5-25).
Drug Therapy	• Oxygen • Blood transfusions • Iron supplements • Dietary and lifestyle changes can help in some cases.	Managed with blood transfusions or exchange transfusions. Oral medications such as Asirox or Deferiprone, or IV medications Deferoxamine.	Parenteral Vitamin B12 or intranasal cyanocobalamin is needed. (Diet changes do not work for their type of anemia), High-dose oral cobalamin and sublingual cobalamin are options for those in	Treatment of folic acid deficiency with replacement therapy. PO medication. For patients who suffer from alcoholism their PO dose will be higher.

		Ascorbic acid supplements may be needed during chelation therapy since they increase the urine excretion of Iron.	whom GI absorption is intact.	
Nursing Management	• Identify underlying cause • Drug therapy • Oral: Ferrous sulfate • IM or IV: iron dextran • Nutritional therapy • Transfusion of packed RBCs.	• Drug therapy • Transfusions • Hematopoietic stem cell transplantation (HSCT)	• Identify underlying cause • Drug therapy	Teach patients the importance of eating food high in folic acid (Green leafy veggies, enriched grain products and breakfast cereals, orange juice, peanuts and avocado).

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Anemia of chronic disease (also called anemia of inflammation) can be caused by cancer, autoimmune and infectious disorders. Underproduction of RBCs and mild shortening of RBC survival. RBCs are usually normocytic, normochromic and hypoproliferative.	The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells.	Occurs because of a sudden hemorrhage.	The effects of chronic blood loss are usually related to the depletion of iron stores and considered an iron deficiency anemia. Destruction or hemolysis of RBCs at a rate that exceeds production.
Clinical Manifestations	Affected individuals may develop a variety of symptoms such as fatigue, paleness of the skin (pallor),	Aplastic anemia can maifest abruptly (over days) or insidiously over weeks to months and can vary to mild	Caused by the body's attempt to maintain an adequate blood volume loss.	• Jaundice • Enlargement of spleen and liver

	lightheadedness, shortness of breath, a fast heartbeat, irritability, chest pain and additional findings.	or severe. General manifestations are fatigue, and dyspnea. As well as cardiovascular and cerebral responses. Patients with low neutropenia (low neutrophils) are at risk for septic shock and death. Thrombocytopenia are predisposed to bleeding.	S&S depends on the amount of blood loss. ● Vasovagal syncope ● Tachycardia with exercise and slight positional hypotension ● central venous pressure and cardiac output below normal at rest, air hunger, rapid thready pulse and cold clammy skin. ● Shock, lactic acidosis and potential death.	
Diagnostic Studies	A bone marrow biopsy so they can test for iron stores and serum iron levels. High iron stores and a low serum iron level may be a sign you have anemia of chronic disease.	Labs: WBC, RBC, hemoglobin and platelet values. Reticulocytes will be low. Serum iron and total	Once plasma volume is replaced, the RBCs become less concentrated and then RBCs, hemoglobin, and hematocrit levels are low and reflect the actual blood loss.	● Monitor kidney functions ● Blood tests and monitor hemoglobin

		iron binding capacity may be high as initial signs of erythropoiesis suppression. Bone marrow biopsies and aspirations.		
Drug Therapy	Blood transfusions are not recommended for long term use. Erythropoietin therapy is used but it is limited because of the increased risk for thromboembolism and death in some patients.	• Immunosuppression therapy, with antithymocyte globulin • Steroids • Cyclophosphamide	• Blood transfusions • IV fluids • Dextran • Hetastarch • Albumin • Crystalloid electrolyte solutions	Ferrous sulfate is the mainstay treatment for treating patients with iron deficiency anemia. It should be continued for about 2 months after correction of the anemia and its etiologic cause in order to replenish body stores of iron.
Nursing Management	• Signs of external bleeding • 12 lead ECG • I&Os • Neuro status • Monitor lab values • Check stools for occult blood • Monitor O2 • Drug therapy medications	Preventions of infection and hemorrhage.	• Replacing blood volume loss to prevent shock • Finding the source the hemorrhage and stopping the blood loss	• Iron supplement • IV fluids • O2 therapy • Blood transfusions

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
	Results from hemolysis of RBCs from extrinsic	Hemochromatosis is an iron	The 2 types of polycythemia are primary

Etiology	factors which include: ● Physical destruction ● Antibody reactions ● Infectious agents and toxins	overload disorder. This is a genetic effect, is an autosomal recessive disorder characterized by increased intestinal iron absorption and as a result increased tissue iron deposition. Can be caused by liver disease or chronic blood transfusions used to treat thalassemia and SCD.	polycythemia (involves WBC, RBC, and platelets with increased production) or polycythemia vera (Genetic) and secondary polycythemia (hypoxia driven or hypoxia independent. The need for o2 may result from high altitude, lung disease, cardiovascular disease, alveolar hypoventilation, detective o2 transport or tissue hypoxia.
Clinical Manifestations	Symptoms include weakness, paleness, jaundice, dark-colored urine, fever, inability to do physical activity, and heart murmur.	Symptoms usually do not develop until after the age of 40 years old in men and after 50 years old in women. Early symptoms include fatigue, arthralgia, impotence, abdominal pain and weight loss. Later symptoms include excess iron accumulates in the liver, pancreas, heart, joints, and endocrine glands which causes diabetes, skin pigment changes, heart problems, arthritis and testicular atrophy.	Often the first manifestations are: ● headache ● vertigo ● dizziness ● tinnitus ● visual changes ● generalized pruritus ● increased basophils ● redness of the hands and feet ● Some patients may have angina ● HF
Diagnostic Studies	Blood tests (Table 30.6)	Laboratory values show a high serum iron, TIBC, and serum ferritin. Testing for known genetic	● High hemoglobin and RBC with microcytosis ● low to normal EPO level

		mutations confirms the diagnosis. Liver biopsy can quantify the amount of iron and establish the degree of organ damage.	• High WBC with basophilia and neutrophils • High platelet count and platelet dysfunction • high leukocyte alkaline phosphatase, uric acid, and cobalamin levels Bone marrow examination in polycythemia vera shows hypercellularity of RBCs , WBCs, and platelets.
Drug Therapy	Aggressive hydration and electrolyte replacement to reduce the risk of kidney injury. Corticosteroids and blood products.	Remove excess iron from the body and minimize any symptoms the patient may have. Iron removal is achieved by removing 500 mL of blood each week for 2-3 years until the iron stores in the body are depleted.	Treatment is to reduce blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment. The goal is to reduce the hematocrit and keep it less than 45%. Myelosuppressive agents such as hydroxyurea, busulfan, and chlorambucil.
Nursing Management	General supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs.	Monitor symptoms of Diabetes, HF	• Assess patients nutritional status. • Begin activities or passive leg exercises

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.