Unit 7: Hematology
Chapter 29 & 30
ONLINE CONTENT (1.5 H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 20, 2023 at 0800. Please be sure to bring a copy to class on March 20, 2023.

Table 1	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	May develop because of inadequate dietary intake, malabsorption, blood loss, or hemolysis	A group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production. Thalassemia is due to an absent or reduced globulin protein.	The most common cause of cobalamin deficiency is pernicious anemia. It is caused by an absence of intrinsic factor. It is a disease of insidious onset. The gastric mucosa is not secreting IF because of either gastric mucosal atrophy or autoimmune destruction of parietal cells.	Also causes megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	May not have any symptoms. As the disease becomes chronic, any of the general manifestations of anemia may develop. Pallor is the most common finding. Glossitis (inflammation of the tongue) is the second most common finding. Another finding is cheilitis (inflammation of the lips). The patient may report headache, paresthesias, and a burning sensation of the tongue.	Patients are often asymptomatic. The patient has mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed color of the skin, and bone marrow hyperplasia. This is a life-threatening disease in which growth, both physical and mental, is often slowed. The patient will present pale and displays other general symptoms of anemia The symptoms develop in childhood by 2 years of age and can cause growth and development deficits.	Develop because of tissue hypoxia. GI manifestations include a sore, red, beefy, and shiny tongue; anorexia, nausea, and vomiting; and abdominal pain. Typical neuromuscular manifestations include weakness, paresthesias of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired thought processes ranging from confusion to dementia.	Common causes include chronic alcoholic, chronic hemodialysis, dietary deficiency, etc. The manifestations are like those of cobalamin deficiency. The disease develops insidiously. The patient's 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, can cause neurologic symptom.
Diagnostic Studies	Laboratory abnormalities characteristic of iron-deficiency	Laboratory abnormalities include decreased Hgb/Hct, increased reticulocytes	The RBCs appear large and have abnormal shapes. This structure contributes to	Serum folate level is low, with a normal serum cobalamin level.

	anemia (Hgb/Hct, MCV, bilirubin, etc). Endoscopy and colonoscopy may detect GI bleeding. A bone marrow biopsy may be done if other tests are inconclusive.	and serum iron and bilirubin.	RBC destruction because the cell membrane is fragile. Serum cobalamin levels are low. Serum folate levels are reviewed. Testing of serum methylmalonic acid and serum homocysteine helps determine the cause of this type of anemia.	
Drug Therapy	Oral iron is a good option because it is inexpensive and convenient. Daily dose should provide 150 to 200 mg of elemental iron. This can be taken in 3 or 4 daily doses, with each tablet or capsule of the iron preparation containing between 50 and 100 mg of iron. Undiluted liquid iron may stain the patient's teeth.	Managed with blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron. Transfusions are given to keep the Hgb level around 10 g/dL, depending on the manifestations. Drugs used include oral deferasirox or deferiprone, or IV or subcutaneous deferoxamine. Ascorbic acid supplements may be needed during chelation therapy since they increase urine excretion of iron.	A typical treatment schedule consists of 1000 mcg/day of cobalamin IM for 2 weeks and then weekly until the hemoglobin is normal and then monthly for life. High-dose oral cobalamin and sublingual cobalamin are options for those whom GI absorption is intact.	Treated with replacement therapy. The usual dosage is 1 mg/day by moth. The patient with malabsorption or chronic alcoholism may need up to 5 mg/day.
Nursing Management	GI side effects may occur, including heartburn, constipation, and diarrhea. Have the patient stay upright for 30 minutes after taking oral forms. If side effects develop, the dose and type of supplement may be adjusted. The patient should start on stool softeners and laxatives, if needed, when started on iron.	Treatment is not needed as the body adapts to the reduction of normal hemoglobin. Because RBCs are sequestered in the enlarged spleen, the patient may need a splenectomy. Hepatic, heart, and lung function are monitored and treated as needed. With proper iron chelation therapy, patients are living longer.	Assess neurologic difficulties that are not fully corrected by replacement therapy. Implement measures to reduce the risk for injury from the decreased sensitivity to heat and pain related to neurologic impairment. Protect the patient from falling, burns, and trauma. In some patients, the neuromuscular complications may not be reversible and physical therapy may be needed.	The patient's symptoms may be attributed to other coexisting problems (cirrhosis, esophageal varices). The duration of treatment depends on the reason for the deficiency. Teach the patient to eat foods high in folic acid.

Table 2	Anemia of Chronic Disease	Aplastic Anemia	Acute Anemia due to blood loss	Chronic Anemia due to blood loss
Etiology	Caused by cancer, autoimmune and infectious disorders, HF, and chronic inflammation.	Disease in which the patient has peripheral blood pancytopenia and hypocellular bone marrow.	Occurs because of sudden hemorrhage. Causes of acute blood loss include trauma, complications of surgery, and conditions or diseases that disrupt vascular integrity.	Similar to those of iron-deficiency anemia.
Clinical Manifestations	Usually develops after 1 or 2 months of disease activity, has an immune basis. Associated with an underproduction of RBCs and mild shortening of RBC survival.	Can manifest abruptly or insidiously over weeks to months. It can vary from mild to severe. Patient may have symptoms caused by suppression of any or all bone marrow elements.	Caused by the body's attempt to maintain an adequate blood volume and meet oxygen requirements.	Effects usually related to the depletion of iron stores and considered an iron-deficiency anemia.
Diagnostic Studies	Other factors may contribute to the anemia. High serum ferritin and increased iron stores distinguished it from iron-deficiency anemia. Normal folate and cobalamin blood levels distinguish it from megaloblastic anemias from folate and cobalamin deficiencies.	Laboratory studies confirm the diagnosis-because it effects all marrow elements, hemoglobin, WBC, and platelet values are decreased. Other RBC indices are generally normal.	The loss of RBCs is not reflected in laboratory data, and values may seem normal or high for 2 to 3 days. Once plasma volume is replaced, the RBC mass is less concentrated.	Laboratory abnormalities (Hgb/Hct, MCV, bilirubin, etc).
Drug Therapy	Best treatment of anemia of chronic	HSCT and immunosuppressive	IV fluids used in emergencies include	Transfusions may be needed.

	disease is to correct the underlying disorder. If severe, blood transfusions may be needed, but they are not recommended fro long-term treatment.	therapy with antithymocyte globulin, steroids, and cyclosporine or cyclophosphamine.	dextan, hetastarchm albumin, and crystalloid electrolyte solutions such as LR.	
Nursing Management	Erythroproietin therapy is used for anemia related to renal disease and cancer and its therapies. It is limited though, because of the increased risk for thromboembolism and death in some patients.	Management is based on identifying and removing the causative agent and providing supportive care until the pancytopenia reveres.	The body needs 2 to 5 days to make more RBCs in response to increased erythropoietin. The nursing care for a patient with anemia resulting from acute blood loss will likely include giving blood products.	Management of chronic blood loss anemia involves identifying the source and stopping the bleeding. Supplemental iron may be needed.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Condition caused by the destruction of hemolysis of RBCs at a rate that exceeds production. In this type of anemia, the RBCs are normal, but external factors are causing damage.	Iron overload disorder. A genetic defect is the most common cause, it may occur with diseases such as sideroblastic anemia. May be caused by liver disease and the chronic blood transfusions used to treat thalassemia and SCD.	Two types of polycythemia are primary and secondary.
Clinical Manifestations	Macrophages, particularly those in the spleen, liver, and bone marrow, destroy RBCs that are old, defective, or moderately damaged. Physical destruction of RBCs results from the exertion of extreme	Symptoms usually do not develop until after age 40 years in men and 50 years in women. Early symptoms are nonspecific and include fatigue, arthralgia, impotence, abdominal pain, and weight loss.	Occur because of the hypertension caused by hypervolemia and hyper viscosity. They are often the first manifestations and include headache, vertigo, dizziness, tinnitus, and visual changes.

	force on the cells. Traumatic events causing disruption of the RBC membrane include hemodialysis, extracorporeal circulation used in bypass, and prosthetic heart valves.		
Diagnostic Studies	Elevated bilirubin levels. Spleen and liver hyperactivity. Kidney function.	Laboratory values show a high serum iron, TIBC, and serum ferritin. Testing for known genetic mutations confirms the diagnosis.	High hemoglobin, RBC count, low to normal EPO level, high WBC with basophilia and neutrophilia, high platelet count and dysfunction.
Drug Therapy	Focused on maintaining renal function. Inlvove general supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs.	Goal of treatment is to remove excess iron from the body and minimize any symptoms the patient may have.	Directed towards reducing blood volume and viscosity and bone marrow activity. Hydration therapy can reduce the blood's viscosity.
Nursing Management	Jaundice is likely because the increased destruction of RBCs causes an elevation in bilirubin levels. Spleen and liver may enlarge because of their hyperactivity, which is related to macrophage phagocytosis of the defective RBCs. May need folate replacement. To suppress the RBC destruction, immunosuppressive agents may be used.	Treatment is same as conventional treatment for these problems. The most common causes of death are cirrhosis, liver failure, liver cancer, and HF. With early diagnosis and treatment, life expectancy is normal.	When acute exacerbations develop, you have several nursing responsibilities. Depending on the agency's policies, you may either assist with or perform the phlebotomy. Assess fluid intake and output during hydration therapy to avoid fluid overload.