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	Inadequate dietary intake, malabsorption, blood loss, or hemolysis	Group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production.	The parietal cells of the gastric mucosa secrete a protein termed IF, this is required for Cobalamin absorption.	Folic acid is needed for DNA synthesis leading to RBC formation and maturations.
Clinical Manifestations	As disease become chronic, any general manifestations of anemia may develop. Pallor is the most common finding.	Often asymptomatic. The pt has mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells). Mild splenomegaly, bronzed color of the skin, and bone marrow hyperplasia.	Develop because of tissue hypoxia. GI manifestations; sore red beefy swollen tongue, anorexia, n/v, abdominal pain. Typical neuromuscular manifestations, weakness, paresthesia of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired thought process ranging from confusion and dementia.	Similar to those of cobalamin deficiency. GI problems may include stomatitis, cheilosis, dysphagia, flatulence and diarrhea.
Diagnostic Studies	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate. Serum test for anti- IF antibodies may be done.	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate
Drug Therapy	Oral Iron	Managed is blood transfusions or exchange transfusions in conjunction with chelating agents that	High dose oral cobalamin and sublingual cobalamin.	Replacement therapy 1mg/day.

		bind to iron. Zinc supplements, folic acid, ascorbic.		
Nursing Management	Treat underlying problem that is causing loss of iron. Increasing dietary iron.	Hematopoietic stem cell transplantation.	Protect patient from falling, burns, and trauma. In neuromuscular complications may not be reversible and PT is needed.	Teach 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 or chronic inflammation. Associated with an underproduction of RBCs and mild shortening of RBC survival.	About 70% of aplastic anemias are due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells.	Causes of acute blood loss include trauma, complications of surgery and conditions or diseases that disrupt vascular integrity.	The effects of chronic blood loss are usually related to depletion of iron stores and considered an iron-deficiency anemia.
Clinical Manifestations	Usually develops after 1-2 month of disease activity, has an immune basis.	Fatigue and dyspnea, as well as cardiovascular and cerebral responses.	The manifestations of anemia from acute blood loss are caused by the body's attempts to maintain an adequate blood volume and meet 02 requirements.	Bleeding, ulcer, hemorrhoids, menstrual and postmenopausal blood loss.
Diagnostic Studies	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate. High serum ferritin and increased iron stores distinguish it from iron-deficiency anemia	Hemoglobin, wbc, and platelets values are decreased.	Lower RBCs, and values may seem normal or high for 2 to 3 days, however, once plasma volume is replaced, the RBC mass is less concentrated. Then, RBC, hemoglobin, and hematocrit levels are low and reflect the actual blood loss.	Lower RBCs, and values may seem normal or high for 2 to 3 days, however, once plasma volume is replaced, the RBC mass is less concentrated. Then, RBC, hemoglobin, and hematocrit levels are low and reflect the actual blood loss.
Drug Therapy	Blood transfusions and erythropoietin therapy	Eltromopag, oral thrombopoietin	IV fluids used in emergencies, include	Supplemental iron may be needed

		receptor agonist, high dose cyclophosphamide, alemtuzumab and androgens	dextan, hetastarch, albumin, and crystalloid electrolytes solutions, such as lactated ringer's solution. Packed red blood cells.	
Nursing Management	The best treatment of anemia of chronic disease is to correct underlying disorder. Blood transfusion may be needed but are not recommended for long-term therapy.	The treatment of choice for adults younger than 55 who do not respond to the immunosuppressive therapy and who have human leukocyte antigen (HLA0 matched, half-match, or unrelated donor is an HSCT.	Carefully monitor for blood loss from various drainage tubes and dressings and implement appropriate actions. The anemia should begin to correct itself once the source of hemorrhage is found, blood loss is controlled and fluid volumes are replaced.	Management of chronic blood loss anemia involves identifying the source and stopping the bleeding.

Table 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	Results from hemolysis of RBCs from extrinsic factors. 1. Physical destruction. 2. Antibody reactions, and 3. Infectious agents and toxins.	Is an iron overload disorder. It may occur with diseases such as sideroblastic anemia. Maybe caused by liver disease and chronic blood transfusions used to treat thalassemia and SCD.	The production and presence of increased numbers of RBCs.
Clinical Manifestations	Weakness, pallor, jaundice, dark-colared urine and fever	Symptoms usually do not develop until after 40 for men and 50 for women. Early symptoms include fatigue, arthralgia, impotence, abdominal pain, and weight loss.	Hypervolemia and hypervisocity. Headache, vertigo, dizziness, tinnitus and visual changes.
Diagnostic Studies	Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum b12, folate	Labs show high serum iron, TIBC and serum ferritin.	High hemoglobin and RBC count with microcytosis, low to normal EPO levels, high WBC count with basophilia and neutrophilia, platelet dysfunction and high leukocyte alkaline phosphate, uric acid, cobalamin levels and high histamine levels.

Drug Therapy	Folate replacement. To suppress RBC destruction, immunosuppression (glucocorticoids or rituximab.	Iron chelating agents may be used. Deferoxamine, which chelates and removes accumulated iron via the kidneys.	Myelosuppressive agents, such as hydroxyurea, busulfan and chlorambucil may be given. Ruxolitinib inhibits the expression of JAK2 mutation is given to patients who have not responded to hydroxyurea. Other therapies include anagrelide which reduce platelet count and inhibit platelet aggregation. Low dose ASA
Nursing Management	Supportive care may include giving corticosteroids and blood products.	Removing excess iron from the body and minimize any symptoms. Iron removal is achieved by removing 500mL of blood each week for 2-3 years until the iron store are depleted.	Treatment is directed at reducing blood volume and viscosity and bone marrow activity. Phlebotomy is mainstay of tx.