Unit 7: Hematology
Chapter 29 & 30

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	Develops because of inadequate dietary intake, malabsorption, blood loss or hemolysis. Decrease hgb/RBC production.	Inadequate production of normal hemoglobin which decreases RBC. Is dye to absent or reduced globulin protein.	The parietal cells of the gastric mucosa secrete a protein termed IF. IF is required for cobalamin absorption.	Causes megaloblastic anemia. Folic acid is needed for DNA synthesis (defective) leading to RBC maturation and formation.
Clinical Manifestations	Palpitations, fatigue, headache, glossitis, pallor, jaundice, tachypnea, dyspnea, orthopnea, vertigo, anorexia, hepatomegaly, ringing in the ears, blurred vision. HF, angina, MI, increased pulse pressures.	Anemia, microcytosis, hypochromia, splenomegaly, bronzed color of the skin, bone marrow hyperplasia.	Tissue hypoxia, GI manifestations include sore, red, beefy, shiny tongue, Anorexia, N/V, abdominal pain, weakness, paresthesia of the feet and hands, ataxia, weakness, dementia, confusion	Tissue hypoxia, GI manifestations, Anorexia, N/V, abdominal pain, weakness, paresthesia of the feet and hands, ataxia, weakness, dementia, confusion, dysphagia, flatulence, cheilosis, diarrhea.
Diagnostic Studies	**Decreased:** Hgb/Hct, MCV, Serum iron, Ferritin, Bilirubin. **Slight increase or decrease** reticulocyte count, **WNL=**Transferrin, b12, and folate. DX: Stool for occult blood.	**Decreased:** Thalassemia major labs, Hgb/Hct, MCV, TIBC, Transferrin, Folate. **Increased**: Serum iron, reticulocyte count, Bilirubin. **WNL=**b12. DX: Stool for occult blood.	**Decreased:** Thalassemia major, hgb/hct, reticulocytes, serum b12. **Increased:** MCV, Serum iron, Ferritin, Bilirubin, Transferrin. **WNL=**TIBC, Folate.	Decreased: Folate, Hgb/Hct, Reticulocytes. Increased: MCV, Serum iron, Transferrin, Ferritin, Bilirubin. WNL=b12. TIBC.
Drug Therapy	Oral iron, vitamin c, IV iron, IM iron.	No specific drug therapies are effective in treating.	High dose oral cobalamin and sublingual whose GI absorption intact. B12 or intranasal cyanocobalamin is needed.	Replacement therapy, 1mg/ day PO. If alcoholic 5mg/day.
Nursing Management	Identify and treat underlying cause. Drug therapy. Ferrous sulfate/gluconate. Iron therapy, nutritional therapy, and Transfusion RBCs.	Blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron. Does not need treatment because body adapts to the reduction of normal hgb.	Reduce risk for injury, sensitivity to heat and pain, protect from falling, burns, trauma, physical therapy.	Identify and treat underlying cause. Drug therapy. Ferrous sulfate/gluconate. Iron therapy, nutritional therapy, and Transfusion RBCs.

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	Caused by decreased number of RBC precursors. cancer, autoimmune infections, HIV, hepatitis, HF, malaria, chronic inflammation.	Decreased number of RBC. Due to autoimmune activity by autoreactive T lymphocytes. Target and destroy the patient's own hematopoietic stem cells.	Blood vessel rupture, trauma, splenic sequestration crisis.	Gastritis, menstrual flow, hemorrhoids.
Clinical Manifestations	Bleeding episodes, Tachycardia, pallor, jaundice, itching, tachypnea, headaches, depression, sensitivity to cold, anorexia, enlarged liver and spleen.	Can manifest abruptly over weeks to months. May vary from mild to severe. Fatigue, dyspnea, cardiovascular and cerebral responses. Thrombocytopenia.	Maintain adequate blood volume and meet O2 requirements.	Maintain adequate blood volume and meet O2 requirements.
Diagnostic Studies	Decreased hgb/hct, MCV, Reticulocytes, Serum iron, TIBC, Transferrin, WNL=Ferritin or high, Bilirubin, b12, Folate.	Decreased hgb/hct, reticulocytes. Increased MCV, serum iron, TIBC. WNL=iron, TIBC, Transferrin, Ferritin, Bilirubin, b12, Folate.	Decreased hgb/hct, MCV. Increased Reticulocytes, WNL=iron, TIBC, Transferrin, Ferritin, Bilirubin, Serum b12, Folate.	Decreased hgb/hct, MCV, Iron, TIBC, Bilirubin. Increased Reticulocytes, WNL= Transferrin, Ferritin, b12, folate.
Drug Therapy	Erythropoietin therapy is used to anemia due to renal disease.	HSCT and immunosuppressive therapy with ant thymocyte globulin, steroids, cyclosporine, or cyclophosphamide.	IV fluids such as dextran, hetastarch, albumin, crystalloid electrolyte solutions such as lactated ringers, blood transfusions	Supplemental Iron.
Nursing Management	Treat underlying disorder. Blood transfusions may be needed.	Preventing complications from infection and hemorrhage.	Prevent blood loss. Fluid blood volumes replaced.	Identifying blood source and stop the bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Hemolysis of RBCs from extrinsic factors. These factors include physical destruction. Antibody reactions, infectious agents, and toxins.	Iron overload disorder. Genetic defect most common cause. Caused by liver diseases and blood transfusions used to treat thalassemia and SCD.	Production and Prescence of increased number of RBCs. The increase can be so great, that blood circulation is impaired because of the increased blood viscosity and volume.
Clinical Manifestations	Bleeding episodes, Tachycardia, pallor, jaundice, itching, tachypnea, headaches, depression, sensitivity to cold, anorexia, enlarged liver and spleen.	Most with gene mutation do not know it. Clinical expression varies and depends on dietary iron and blood loss. Fatigue, arthralgia, impotence, abdominal pain, and weight loss. Later cirrhosis. Diabetes, bronzing, heart problems, arthritis and testicular atrophy.	Headache, dizziness, tinnitus, visual changes, itching, paresthesia's, angina, HF, intermittent cloudification, thrombophlebitis, redness of hands and feet.
Diagnostic Studies	Decreased hgb/hct/ Increased MCV, Reticulocytes, serum iron, Ferritin, Bilirubin. WNL=Transferrin, b12, folate.	High serum iron. TIBC, serum ferritin. Testing for genetic mutations confirms the diagnosis. Liver biopsy can quantify amount of iron and see organ damage.	High hgb and RBCs low to normal EPO, high WBCs count with basophilia and neutrophilia. High platelet count, high leukocytes alkaline phosphatase, uric acid, cobalamin levels and high histamine levels.
Drug Therapy	Folate replacement Corticosteroids Blood products	Iron chelating agents are used. Deferoxamine. Deferasirox.	Myelosuppressive agents such as hydroxyurea, busulfan and chlorambucil may be given. Low dose aspirin and allopurinol.
Nursing Management	General supportive care until the causative agents can be eliminated or at least made less injurious to the RBCs. Aggressive hydration and electrolyte may be needed.	Managing diabetes, HF. Avoid vitamin C, iron supplements, uncooked seafood. Remove excess iron from the body and minimize any symptoms the patient has.	Reducing blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment. Reduce hct to below 45%