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.	A group of disease involving inadequate production of normal hemoglobin, which decreases RBC production. Due to an absent or reduced globulin protein. There is a genetic link.	It is caused by an absence of intrinsic factor; Intrinsic factor is required for cobalamin extrinsic factor absorption.	Also causes megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	Early the patient many do not have symptoms, Pallor is most common, inflammation of the tongue is the second most common and inflammation of the lips, headaches paresthesia's and burning sensation of the tongue.	Minor is often asymptomatic microcytosis (small cells) and hypochromia (pale cells) mild splenomegaly, bronzed color skin and bone marrow hyperplasia. Major is life threatening patient is pale developmental deficits, pronounced splenomegaly hepatomegaly and cardiomyopathy, chronic bone marrow hyperplasia. Cardiac complications form iron overload, lung disease and hypertension also contribute to early death.	Related to cobalamin deficiency develop because of tissue hypoxia. GI manifestation include a sore, red, beefy, and shiny tongue, anorexia, nauseam and vomiting, and abdominal pains, Neuromuscular manifestation include weakness, paresthesia's of the feet and hands, ataxia, muscle weakness and impaired thought process ranging from confusion to dementia. May take several months for manifestations to develop	The disease similar to cobalamin deficiency this develops insidiously. The patients' symptoms may be attributed to other coexisting problems such as cirrhosis esophageal varices, stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Thiamine deficiency is often present, with folate deficiency, can cause neurologic symptoms.
Diagnostic Studies	Hemoglobin, Hematocrit, RBC count, reticulocyte	History and physical lab work HGB/HCT MCV,	Labs such as Hgb/HCT, MCV Reticulocytes,	Labs such as Hgb/HCT, MCV Reticulocytes,

Diagnostic Studies	count, serum iron, serum ferritin, serum transferrin, occult blood, endoscopy and colonoscopy, bone marrow biopsy.	Reticulocytes, Serum Iron, TIBC, Transferrin, Ferrin, Bilirubin, Serum B12, Folate	Serum Iron. TIBC, Transferrin, Ferritin, Bilirubin, Serum B12, Folate.	Serum Iron. TIBC, Transferrin, Ferritin, Bilirubin, Serum B12, Folate.
Drug Therapy	Iron supplement daily does should be 150-200 mg can be taken 3 or 4 daily doses,	No specific drug or diet therapies are effective in treating, Minor does not need treatment. Major is treated by blood transfusions or exchange transfusion with chelating agents that bind to iron. Oral deferasirox or deferiprone or IV or subcutaneous deferoxamine, Ascorbic acid	Parenteral vitamin B12, or intranasal cyanocobalamin. High dose of oral cobalamin and sublingual cobalamin are options for those in whom GI absorption is intact.	Replacement therapy usual dose is 1 m, with malabsorption of chronic alcoholism may need up to 5 mg/day/by mouth.
Nursing Management	Treat the underlying problem that is causing iron loss or reduced intake, such as malnutrition, alcoholism, or poor absorption of iron.	Monitor evidence of cyanosis, dietician referral, monitor oxygen pulse ox, monitor signs of hypoxia, provide oxygen therapy monitor vitals. Observe any restlessness and confusion.	Implement measures to reduce the risk for injury, from the decreased sensitivity to heat and pain related to neurologic impairment. Protect patient from falling, burns and trauma.	Teach the patient d\ to eat foods high in folic acid. Maintain drug therapy

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	Can be caused by cancer, autoimmune and infectious disorders, HIV, hepatitis, malaria, HF, or chronic inflammation. Bleeding episodes can contribute to anemia of chronic disease. Associated with an under production of RBC and mild shortening of RBC	70% due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells. Other anemias may be acquired from toxic injury to bone marrow stem cells or result from	Due to sudden blood loss of hemorrhage including trauma, complication of surgery and conditions or diseases that disrupts vascular integrity.	Similar to those of iron deficiency anemia. Usually related to the depletion of iron stores and considered an iron deficiency anemia.

	survival.	inherited stem cell defect.		
Clinical Manifestations	Anemia usually develops after 1 to 2 months of disease activity, The cytokines released in these conditions particularly interleukin cause an increased uptake and retention of iron within macrophages, this leads to a diversion of iron from circulation into storages sites with subsequent limited iron available	Can manifest abruptly over days or insidious over weeks. Can very from mild to severe. Patient may have symptoms caused by suppression of any or all bone marrow elements. Fatigue, and dyspnea and well as cardiovascular and cerebral responses, May be susceptible to infections and at risk for septic shock and death	Tachycardia with exercise and slight postural hypotension, Postural hypotension, and tachycardia with exercise. BP central venous pressure and cardiac output below normal at rest air hunger rapid thready pulse and cold clammy skin, shock, Lactic acidosis, possible death.	Bleeding ulcers, hemorrhoids, menstrual and postmenopausal blood loss.
Diagnostic Studies	Folate and cobalamin blood levels.	Bone marrow elements, hemoglobin, WBC RBC, TIBC	RBC, Hgb, Hct.	Hgb, Hct. RBC including morphology, Reticulocyte count, serum iron, serum ferritin, serum transferrin, total iron binding capacity, stool for occult blood.
Drug Therapy	Blood transfusions may be needed but not recommended for long term treatment. Erythropoietin therapy is used for anemia related to renal disease. And cancer ant its therapy.	Steroids. And cyclosporine, immunosuppressive therapy with antithymocyte globulin. Promacta and oral thrombopoietin receptor agonist.	IV fluids in emergency dextran, hetastarch, albumin, crystalloid electrolyte, blood transfusion packed RBC,	Iron supplement and vitamin c for absorption.
Nursing Management	Treat underlining causes, education on drug therapy management, nutritional therapy, Education on	Providing support for patient and family, Educate on drug therapy. Preventing complications.	Monitor blood loss. Replace blood that was lost. Finding source of hemorrhage. Vital signs	Identify the source. Stop the bleeding Education on iron supplements and follow up appointments.

	symptoms and when to come back to the hospital.	Monitor signs and symptom.		Monitor Vitals.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	The RBC are normal but external factors are causing damage Macrophages. Particularly those in the spleen, liver, and bone marrow,	Is an overload disorder. Although a genetic defect is the most common cause, may occur with diseases such as sideroblastic anemia, and maybe caused by liver damage, and chronic blood transfusions used to treat thalassemia and SCD/	Is the production and presence of increased numbers of RBC's. The RBC's can be so great that blood circulation is impaired because of the increased blood viscosity and volume. Some people have a genetic link.
Clinical Manifestations	Jaundice is likely because the increased destruction of RBC, elevation of bilirubin, The spleen and liver are enlarged,	Symptoms usually do not develop until after the age of 40 in men and 50 in women, fatigue, arthralgia, impotence abdominal pain and wight loss, later liver enlargement and cirrhosis, bronzing of the skin heart problems, arthritis, and testicular atrophy.	Hypertension caused by hypervolemia, and hyper viscosity, headache, vertigo, dizziness, tinnitus, and visual changes, generalized pruritus often exacerbated by a hot bath, painful and burning of hands and feet, angina, HF, intermittent claudication and thrombophlebitis, impaired blood flow, thrombosis and tissue hypoxia from hypervolemia and hyper viscosity, inadequate platelet function GI bleed bruising, petechiae.
Diagnostic Studies	Bone marrow tests, genetic tests, RBC, Hgb and Hct, Serum iron, Serum ferritin, Serum transferrin, total iron binding capacity, stool for occult.	Serum iron, TIBC and serum ferritin, genetic mutations confirm diagnosis. Liver biopsy can quantify the amount of iron and establish the degree of organ damage.	Hemoglobin, RBC, microcytosis, WBC with basophilia and neutrophilia, platelet count, leukocyte, uric acid, and cobalamin levels, histamine levels, bone marrow bx.
Drug Therapy	Corticosteroid, medicines, rituximab, and cyclosporine,	Remove excess iron from the body, Iron chelating agents deferoxamine deferasirox, deferiprone. Avoid vitamin C and iron supplements,	Myelosuppressive agents, Hydroxyurea, Busulfan, Chlorambucil, Ruxolitinib, Anagrelide, Allopurinol to reduce gouty attacks
	Dietician consult, assess	Avoid uncooked seafood	Remove excess fluids from

Nursing Management	fatigue, pain management, vitals,	and iron rich foods. Education on medication and disease process	patient. Intake and output. Vitals signs, Educate patient about their medications,