Unit 7: Hematology
Chapter 33 & 34
ONLINE CONTENT (2H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 17, 2025 at 0800. Please be sure to bring a copy to class on March 17, 2025.

Table 1	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	A deficiency in the number of erythrocytes, the quality of hemoglobin, and/or volume of packed RBC	A group of diseases involving inadequate production of normal Hgb, which increases RBC production.	Caused by absence of intrinsic factor. Normally, the parietal cells of the gastric mucosa secrete IF.	Can cause megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	Mild anemia (Hgb 10-12 g/dL to moderate anemia (Hgb 6-10g/dL)	Often asymptomatic, mild to moderate anemia with microcytosis	Sore, red, beefy, and shiny tongue, paresthesia of the feet, muscle weakness etc.	Develops insidiously, stomatitis, cheilosis, dysphagia, flatulence.
Diagnostic Studies	Labs: Hgb/Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate	Labs: Hgb/Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate	Labs: Hgb/Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate	Labs: Hgb/Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate
Drug Therapy	Oral iron	Does not need treated as the body adapts to the reduction of normal Hgb.Oral asirox, deferiprone, or IV/SQ deferoxamine	1000mcg/day of cobalamin IM for 2 weeks, then weekly until the Hgb is normal, and then monthly for life	Replacement therapy, usually dosage is 1 to 5 mg/day.
Nursing Management	Encourage alternate rest and activity as correcting the cause is the goal	Watch Hgb as blood transfusion is needed for levels less than 7g/dL	Protect the patient from falling, burns, and trauma, may need physical therapy.	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	Underproduction on RBC and mild shortening of RBC survival.	The patient has peripheral blood pancytopenia and hypocellular bone marrow.	Sudden bleeding causes of acute blood loss include trauma, surgery complications. And problems that disrupt vascular	The effects of chronic blood loss are usually due to depleted iron stores.

			integrity.	
Clinical Manifestations	Develops 1-2 months after disease activity. Causes such as cancer and HIV.	Aplastic anemia can manifest abruptly (over days) or insidiously over weeks to months.	Acute blood loss is caused by the body's attempts to maintain an adequate blood volume and meet O2 requirements.	General manifestations of anemia with some specific manifestations, the spleen and liver may be enlarged.
Diagnostic Studies	High serum ferritin and increased iron stores distinguish it from iron deficiency anemia.	Hgb, WBC, and platelet values, iron, RBC, TIBC, bone marrow biopsy, and aspiration.	Labs do not reflect the RBC loss, plasma, RBC, Hgb, Hct.	RBC, Hgb
Drug Therapy	Treatment is to correct underlying problem and sometimes a blood transfusion is needed.	Immunosuppressive therapy and antihymocyte globulin and cyclosporine	Blood transfusions (whole blood, platelets, plasma, and cryoprecipitate)	Iron supplements
Nursing Management	Must be recognized and distinguished from anemia of other causes.	Management is based on identifying and removing the causative agent and proving supportive care until the pancytopenia.	Replacing blood volume to prevent shock, promoting coagulation to prevent further bleeding, and finding the source of the bleeding and stopping the blood loss	Maintain renal function

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Results from hemolysis of RBC from extrinsic factors.	Iron overload disorder characterized by increased intestinal iron absorption.	Production and presence of increased numbers of RBC. Primary or secondary.
Clinical Manifestations	Traumatic events that disrupt the RBC membrane include hemodialysis, extracorporeal circulation used in cardiopulmonary bypass, and prosthetic heart valves.	Symptoms usually do not develop until after age 40 years in men and 50 years in women.	Circulatory manifestations occur due to the hypertension caused by hypervolemia and hyperviscocity.
Diagnostic	Labs: Hgb/Hct, MCV,	Iron, TIBC, serum ferritin,	Hgb. Hct, RBC, bone

Studies	reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate	genetic mutations, MRI, cardiac iron, liver biopsy	marrow examination, WBC, platelets, and JAK2 mutation.
Drug Therapy	Folate replacement and immunosuppressants	Iron-chelating drugs	Myelosuppressive agents
Nursing Management	Aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by Hgb shock.	Remove excess iron from the body and minimize any symptoms the patient may have.	Assess the patients nutrition statis, inadequate food intake can result from GI symptoms of fullness, pain, and dyspepsia.

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.