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	It may develop due to inadequate dietary intake, malabsorption, blood loss, or hemolysis.	A group of diseases involving inadequate production of normal hemoglobin, that decreases RBC production. It is due to an absent or reduced globulin protein. Hemolysis occurs as most erythroblasts are destroyed by mononuclear phagocytes in the marrow.	In pernicious anemia the gastric mucosa is not secreting IF which is needed for cobalamin absorption. This can occur in patients who have had a gastrectomy, or gastric bypass, small bowel resection involving the ileum, crohn's disease, ileitis, celiacs disease, diverticula of the small intestine, and chronic atrophic gastritis.	Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	In early stages the patient may not have symptoms. As the disease becomes chronic they may have general manifestations such as pallor (the most common), glossitis, cheilitis, headache, paresthesia, and burning sensation of the tongue all due to lack of blood in the tissues.	Those with thalassemia minor are often asymptomatic, they will have minor anemia with microcytosis and hypochromia, mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. It is a major life threatening disease in which both physical and mental growth are slowed.	Sore, red, beefy, and shiny tongue: anorexia, nausea, vomiting, and abdominal pain. Neuromuscular symptoms are weakness, paresthesia of the feet and hands, ataxia, muscle weakness, and impaired thought processes.	Sore, red, beefy, and shiny tongue: anorexia, nausea, vomiting, and abdominal pain, stomatitis, chelosis, dysphagia, flautulence, diarrhea. Neuromuscular symptoms are weakness, paresthesia of the feet and hands, ataxia, muscle weakness, and impaired thought processes.
	Normal lab draws, stool occult blood	Normal lab draws,	RBC shape and size, serum folate levels,	Normal lab draws, low serum folate

Diagnostic Studies	tests, endoscopy, colonoscopy, and bone marrow biopsy.		a serum test for anti-IF- antibodies, serum methylmalonic acid, serum, homocysteine levels.	levels with normal cobalamin levels.
Drug Therapy	Oral iron supplements, or parenteral iron. The parenteral iron is for those who have malabsorption, intolerance of oral iron, or a need for iron beyond oral limits.	Blood transfusions or exchange transfusions in conjunction with chelating agents to bind to iron. Drugs such as asirox, deferiprone, or IV or sub Q deforoxamine. Zinc supplements and folic acid.	Parenteral vitamin B12, intranasal cyanocobalamin. If the Gi absorption is intact they may take oral or sublingual cobalamin.	Folic acid replacement therapy. 1mg/ day is the normal dosage, those with chronic alcoholism may need up to 5mg/ day.
Nursing Management	You need to reassess Hgb and RBC count to evaluate the response to therapy. Ensure stressing importance of adherence with dietary and drug therapy. Iron therapy must be taken for 2-3 months after the return of normal Hgb. Monitor patients for potential liver problems who receive lifelong iron treatment.	Monitor BP, ensure medication adherence, monitor for signs of pulmonary hypertension. Skin assessments for jaundice.	Implement measures to reduce risk for injury, measures for sensitivity to heat and pain, protect the patient from falls, burns, and trauma. Possible physical therapy may also be needed.	Teach the patient about foods high in folic acid, and ensure reduced risk for injury.

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	About 70% are due to autoimmune activity by	Occurs due to sudden hemorrhage. Causes include:	The sources are similar to those of iron deficiency such

	infectious disorders, HF, or chronic inflammation. Bleeding episodes can contribute to anemia of chronic disease.	autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells.	trauma, complications of surgery, and conditions or diseases that disrupt vascular integrity. 2 concerns are hypovolemic shock from reduced total blood volume and The number of RBC's available.	as bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss.
Clinical Manifestations	Causes an underproduction of RBC's and mild shortening of RBC survival. The RBC's typically appear as normocytic, normochromic, and hypoproliferative.	Can manifest abruptly over days or insidiously over weeks to months. It can vary from mild to severe. General manifestations of anemia such as, fatigue, dyspnea, as well as cardiovascular and cerebral responses. If they are neutropenic, they may be at risk for infections and septic shock. They also have a predisposition to bleeding.	The manifestations of anemia from acute blood loss are caused by the body's attempts to maintain and adequate blood volume and meet O2 requirements.	The effects are usually related to the depletion of iron stores and considered an iron-deficiency anemia.
Diagnostic Studies	High serum ferritin, and increased iron stores distinguish this anemia from iron-deficiency. Normal folate and cobalamin distinguish this anemia from folate and cobalamin deficiency.	Laboratory studies confirm the diagnosis. Aplastic anemia affects all marrow elements, hemoglobin, WBC, and platelet values. You can also do various iron studies, bone marrow biopsies, and pathology studies.	The loss of RBC's is not reflected in the laboratory data. However, once the plasma volume is replaced, the RBC mass is less concentrated. Then RBC, hemoglobin, and hematocrit levels are low and reflect the actual blood loss.	Iron levels, RBC levels, monitoring vital signs, and monitoring blood loss.
Drug Therapy	Correct the underlying disorder. Blood transfusions may be used if blood loss is severe. Erythropoietin	Identify and remove the causative agent. There is a poor prognosis, they will need medical management as well	IV fluids used in emergencies are dextran, hetastarch, albumin, and crystalloid electrolyte	Iron may be needed.

	therapy is used for anemia related to renal disease, cancer.	as immunosuppressants . HLA can also help those with this type of anemia.	solutions, as well as lactated ringers. The infusion amount depends of the volume lost.	
Nursing Management	Monitor vital signs, monitor blood work, ensure med adherence as well as frequent PCP visits.	Emotional support of the patient, monitoring blood work, monitor vital signs, ensure medication adherence, ensure isolation from other sources of illness and maintaining regular PCP visits.	Replacing blood volume to prevent shock and finding the source of the bleeding and stopping it.	Identifying the source and stopping the bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Results from hemolysis of RBC's from an extrinsic factor. These factors include 1.) physical destruction, 2.) antibody reactions, 3.) infectious agents and toxins.	Is an iron overload disorder. Genetic effect is the most common cause however, it can occur with diseases such as sideroblastic anemia. Can also be caused by liver disease and the chronic blood transfusions used to treat thalassemia and SCD.	Production and presence of increased RBC's. The number can be so increased if impairs blood circulations causing hyper viscosity and hypervolemia.
Clinical Manifestations	Are similar to those of other anemias. More severe cases and cause DIC or TTP.	Total body iron may exceed concentrations of 50 g. symptoms include fatigue, arthralgia, impotence, abdominal pain, and weight loss. Later they may have liver cirrhosis, an enlarged liver, diabetes, skin pigment changes, heart problems, arthritis, and testicular atrophy.	Hypertension, headache, dizziness, vertigo, tinnitus, and visual changes, paresthesia, general pruritis.
Diagnostic Studies	Laboratory findings. And bloodwork.	Iron levels, TIBC, and serum ferritin. Testing for genetic mutations confirm the diagnosis. Liver biopsy can quantify the amount or iron and the amount of organ damage.	High hemoglobin and RBC with microcytosis, low to normal EPO level, high WBC with basophilia and neutrophilia, high platelet count, high leukocyte alkaline phosphate.
	Corticosteroids and blood products may be	Removing excess iron from the body. Iron removal by	Reducing blood volume and viscosity and bone

Drug Therapy	given. Folate replacement therapy, and monoclonal antibodies.	removing 500 mL of blood each week for 2-3 years. Iron chelating agents, deferoxamine,	marrow activity. 300-500 mL of blood will be removed every few days until acceptable levels are reached. They may also need hydration therapy. Myelosuppressive agents like hydroxyurea, busulfan, and chlorambucil.
Nursing Management	General supportive care, providing medications, and ensuring decreased risk for injury.	Managing the organ involvement, early diagnosis, treating conditions caused by this disorder.	Monitor strict I's and O's, teach the patient about side effects of drugs like the myelosuppressive agents. Assess nutritional status and assess for complications of the polycythemia.