Unit 7: Hematology
Chapters 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	- May develop because of inadequate dietary intake, malabsorption, blood loss, or hemolysis.	- A group of diseases involving inadequate production of normal hemoglobin decreases red blood cell production.	- A deficiency of vitamin B12 is caused by the lack of secretion of the protein IF.	- Deficiency of folic acid due to chronic alcoholism, chronic hemodialysis, dietary deficiency, drug interference, increased requirement, and malabsorption syndromes.
Clinical Manifestations	- In earlier stages, there may not be symptoms. As it progresses symptoms such as pallor, glossitis, cheilitis, headache, paresthesia, and a burning sensation of the tongue.	- With thalassemia minor, the patient will be most likely asymptomatic. With thalassemia major physical and mental growth are slow. The patient will display symptoms of anemia.	- General manifestations of anemia along with GI manifestations include a sore, red, beefy, and shiny tongue; anorexia, nausea, abdominal pain, and vomiting.	- Similar to cobalamin deficiency.
Diagnostic Studies	- Decreased hemoglobin and hematocrit, decreased MCV, decreased serum iron, decreased ferritin, and/or decreased bilirubin.	- CBC and hemoglobin test.	- Serum folate levels are reviewed. Upper GI endoscopy and biopsy of the gastric mucosa.	- Serum folate level.
Drug Therapy	- Oral iron supplements Are the most common and effective therapy. However,	- Thalassemia major is managed by blood transfusions	- Cobalamin administration via oral and sublingual routes	- Replacement therapy via the oral route.

	paternal iron may need to be used.	because there is no specific drug or diet that is effective.		
Nursing Management	- The main goal is to treat the underlying problem that causes iron deficiency. We also need to replace the iron.	- Hepatic, heart, and lung functions need to be monitored during and after treatment.	- Assess for neurological difficulties, implement measures to reduce the risk of injury, and protect the patient from falling, burns, and trauma.	- Teach the patient to eat foods high in folic acid as well as do the same measures as you would for anemia.

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	- The underproduction of RBCs and mild shortening of RBC survival.	- Caused by autoimmune activity by autoreactive T lymphocytes. The patient's own hematopoietic system is targeted.	- Blood loss caused by sudden hemorrhage.	- Long-term effects of uncontrolled blood loss.
Clinical Manifestations	- Mild anemia with more severe symptoms if the underlying disorder is not treated.	- General manifestations of anemia as well as cardiovascular and cerebral responses.	- Normal manifestations of anemia will vary depending on the severity of blood loss.	- Same symptoms as acute anemia due to blood loss, but maybe more severe.
Diagnostic Studies	- Test for high serum ferritin and increased iron stores.	- Laboratory studies.	- Once the plasma volume is replaced RBC, hemoglobin, and hematocrit levels are low on CBC.	- Same as acute anemia due to blood loss.
Drug Therapy	- Correct the underlying disorder. May need blood transfusions.	- HSCT and immunosuppressive therapy. For those who do not respond to immunosuppressive therapy, a transfusion may be done.	- Replacing the blood volume.	- Same as acute anemia due to blood loss.
Nursing	- Administer blood	- Interventions aimed at preventing	- Finding the source of the	- Same as acute anemia due to

Management	transfusions as needed.	complications from infection and hemorrhage.	hemorrhage and stopping blood loss. Replacement of lost blood.	blood loss.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	- Results from hemolysis of RBCs from extrinsic factors. These include physical destruction, antibody reactions, and infectious agents and toxins.	- Iron overload disorder may be caused by sideroblastic anemia, liver disease, and chronic blood transfusions used to treat thalassemia and SCD.	- Primary polycythemia is caused by an increased production in RBCs, WBCs, and platelets. Secondary polycythemia is either hypoxia-driven or hypoxia independent and causes EPO production in the kidneys, which in turn stimulates RBC production.
Clinical Manifestations	- Fever, weakness, dizziness, confusion, dark-colored urine, jaundice, and abnormal paleness.	- Early symptoms are nonspecific such as fatigue, impotence, and abdominal pain. Later symptoms can include liver enlargement and cirrhosis.	- Headache, Vertigo, dizziness, tinnitus, visual changes, general pruritis, angina, heart failure, intermittent claudication, and thrombophlebitis.
Diagnostic Studies	- CBC.	- Laboratory values will show a high serum iron, TIBC, in serum ferritin.	- High hemoglobin and RBC count as well as low to normal EPO levels can be detected on a CBC.
Drug Therapy	- Folate replacement.	- Iron chelating agents.	- Myelosuppressive agents.
Nursing Management	- General supportive care until the causative agent can be eliminated.	- Remove excess iron from the body and minimize symptoms.	- Assist or perform phlebotomy, assessed fluid intake and output, administer myelosuppressive agents, and teach the patient about the side effects of the drugs.