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

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 18, 2024 at 0800. Please be sure to bring a copy to class on March 18, 2024.

Table 1	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	Develops from inadequate diet intake, malabsorption, blood loss, or hemolysis.	Group of diseases involving inadequate production of normal Hgb, which decreases RBC production. This happens from a reduced globulin protein. This is commonly found in origins near the Mediterranean sea.	This happens if we do not have IF and do not absorb Cobalamin. This is common with patients who have had GI surgery, small bowel resection, Crohn's disease, or Celiac Disease.	Folic acid is needed for DNA synthesis, leading to RBC formation and maturation.
Clinical Manifestations	Early course usually doesn't have symptoms. As it becomes chronic pallor, glossitis, cheilitis, headache, and a burning sensation of the tongue is common.	If it is minor they are often asymptomatic. If mild-moderate anemia bronzed skin color and bone marrow hyperplasia is common, along with jaundice.	Sore, red, beefy, and shiny tongue; anorexia, nausea and vomiting; along with abdominal pain. Some neuromuscular issues can happen too such as: weakness, paresthesia of the feet and hands.	Stomatitis, cheilosis, dysphagia, flatulence, and diarrhea.
Diagnostic Studies	Stool Occult blood test, endoscopy, colonoscopy, hgb, hct, rbc count, iron level. If inconclusive sometimes a bone marrow biopsy is done.	Bone marrow biopsy can be done to determine.	Serum cobalamin levels will be low. Normal serum folate levels.	Serum folate will be low (less than 5). Normal serum cobalamin level.
Drug Therapy	Oral Iron supplement is given. When giving iron daily dose should be 100-200 mg. It should be taken an hour before meals.	Blood transfusions, chelating agents such as oral deferasirox, deferiprone, or IV or subcutaneous deferoxamine.	Cobalamin administration, parenteral vitamin B12, intranasal cyanocobalamin, topical treatment for 2 weeks, then	Replacement therapy, with a usual dosage of 1-5 mg/day by mouth.

	(Ferrous Sulfate)		weekly until Hgb is normal, then monthly for life.	
Nursing Management	Identify and treat underlying cause, use drug therapy, or a packed RBC transfusion.	Use drug therapies along with blood transfusions if hgb drops less than 7.	Assess for neurologic problems that are not corrected by replacement therapy. Reduce the risk for injury from sensitivity to heat and pain. Protect patient from burns and trauma.	Teach 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 of RBCs and mild shortening of RBC survival. Commonly caused by cancer, autoimmune and infectious disorders, and chronic inflammation.	Due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells. Other causes include toxic injury to bone marrow stem cells or an inherited stem cell defect.	Due to trauma, surgery complications, and problems that disrupt vascular integrity. The amount of RBC's available to carry O2 is significantly decreased.	Caused by bleeding ulcers, hemorrhoids, menstrual and postmenopausal blood loss. It is due to depletion in iron stores.
Clinical Manifestations	Fatigue, pallor, lightheadedness, chest pain.	Fatigue, dyspnea, neutropenia, petechiae, bruising, or nosebleeds.	Symptoms depend on blood loss. If 10% usually no symptoms or vasovagal syncope. If 20% increased heart rate with exercise and slight hypotension. If 30% postural hypotension. If 40% BP along with cardiac output is below normal at rest. If 50% shock, lactic acidosis.	Symptoms can vary but very similar to those of acute anemia.
Diagnostic Studies	-High serum ferritin and increased iron stores. -Normal folate and	-Lab diagnostics: Hgb, WBC, platelet count will be decreased, reticulocyte count	At first: normal or high for 2-3 days. After plasma volume is replaced, RBCs, Hgb, and hct	Reduced RBCs, hgb, and hct.

	cobalamin blood levels	will be low -Serum iron and TIBC may be high initially along with decreased RBCs -Bone marrow aspiration is definitive	become low and reflect the blood loss.	
Drug Therapy	Blood transfusions if severe. Not recommended for long-term treatment. EPO therapy is used for anemia if caused by renal disease.	- Immunosuppressive therapy with antithymocyte globulin and cyclosporine -Eltrombopag (increases platelet count)	Blood transfusions can be used depending on the severity. If large volume is lost, whole blood, platelets, and plasma can be given. IV fluids can also be used to replace fluids. Lastly colloids can be used to pull fluids into the vascular space.	Blood transfusions are often used. Iron supplements are also used usually.
Nursing Management	Correct the underlying problem.	Management is based in identifying and removing the causative agent and providing supportive care until the pancytopenia resolves. Also preventing complications from infection and bleeding.	Concerned with replacing blood volume to prevent shock, promoting coagulation to prevent further bleeding, and finding the source of the bleeding and stopping the blood loss.	Management involves identifying the source and stopping the bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Hemolysis of RBCs from extrinsic factors. Such as physical destruction, antibody reactions, and infectious agents and toxins., caused by extreme force on RBCs.	Overload disorder characterized by increased intestinal iron absorption. A genetic defect is normally the cause.	-Primary involves increased production of RBCs, WBCs, and platelets. Leading to enhanced blood viscosity and volume. -Secondary is caused by hypoxia or hypoxia independent. Hypoxia stimulates the kidneys to make EPO and stimulating RBC production.
Clinical	Tiredness, weakness enlarged spleen. Purpura lesions.	Symptoms don't develop until after age 40 in men and 50 in women. Early	First symptoms include: headache, vertigo, dizziness, tinnitus, and

Manifestations		symptoms are nonspecific: fatigue, arthralgia, abdominal pain, and weight loss. Later symptoms: bronzing of the skin, heart problems, arthritis, enlarged spleen and liver.	visual changes. A striking symptom is generalized itching exacerbated by a hot bath.
Diagnostic Studies	Blood tests examining platelet, RBC count.	-High serum iron, TIBC, and serum ferritin -Confirmed by genetic testing -MRI measures liver and cardiac iron -Liver biopsy shows amount of iron and establishes degree of organ damage	1) high hgb, hct, and rbc mass 2) bone marrow shows hypercellularity of RBCs, WBCs, and platelets 3) presence of JAK2, Jak2 exon 12 mutation 4) low EPO levels
Drug Therapy	If emergency: give aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by hgb clogging the kidney tubules and shock. Supportive care also says to give corticosteroids.	Iron chelation drugs, Deferoxamine chelates removes iron from the kidneys.	-Hydration therapy reduces the blood viscosity and prevents clotting. -Myelosuppressive agents are also used
Nursing Management	General supportive care until causative agent is eliminated or made less injurious to RBCs.	Manage problems by organ involvement, and doing normal treatment for the problems.	-Assess intake and output during hydration therapy to avoid fluid overload or fluid deficit -Assess the patient's nutrition status -Begin activities and drug therapy to decrease the risk of thrombus formation

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.