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

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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	Deficiency in the number of RBCs, quality and quantity of, and volume of the packed RBCs. May develop from inadequate diet intake, malabsorption, blood loss, or hemolysis.	Group of diseases involving inadequate production of Hgb, leading to decreased RBC production. Due to absent or reduced globulin protein.	Caused by an absence of intrinsic factors. This stops the absorption of cobalamin, the gastric mucosa does not secrete IF because of gastric mucosal atrophy or autoimmune destruction of parietal cells.	Decrease of folic acid available to allot to DNA synthesis of RBC formation and maturation.
Clinical Manifestations	Palpitations, blurred vision (severe), hepatomegaly, swallowing problems, sore mouth (all severe), mild fatigue, bone pain (severe), exertional dyspnea, headache, vertigo (severe), pallor, jaundice, itching (all severe).	Mostly asymptomatic, may have mild-moderate anemia with small, pale cells, mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Jaundice and pale. Hepatomegaly and cardiomyopathy.	Tissue hypoxia, sore/red/beefy/shiny tongue, anorexia, N/V, abdominal pain, weakness, paresthesia of feet and hands, reduced position senses, ataxia, and impaired cognition.	S/s could be part of existing problems. Stomatitis, cheilosis, dysphagia, flatulence, diarrhea, neurologic problems.
Diagnostic Studies	Stool occult test, endoscopy, colonoscopy, bone marrow biopsy, CBC, blood smear.	Hgb/Hct, MCV, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, Serum b12, Folate	Large RBCs w/ abnormal shapes, low serum cobalamin. Test for anti-IF antibodies. Endoscopy, gastric mucosa biopsy.	Hgb/Hct, MCV, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, Serum b12, Folate, low serum folate level, normal serum cobalamin.
Drug Therapy	Educate on increase of foods in iron, daily multivitamin, daily iron supplement. IV supplements or RBC transfusion (severe).	Major blood transfusions or exchange transfusions w/ chelating agents the bind to iron. Oral deferasirox or	Parenteral vitamin b12, intranasal cyanocobalamin, 1000mcg/day of cobalamin IM for two weeks, then monthly for life.	Folic acid replacement therapy with 1-5mg/day PO.

		deferiprone, IV subcutaneous deferoxamine, or Reblozyl subcutaneously.		
Nursing Management	Goal is to find underlying cause of anemia.	Monitor liver, heart, and lung function. HSCT is only cure.	Assess for neurologic problems, educate patient on risk reduction strategies like falls, burns, and trauma. Physical therapy.	Teach pt. 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	Assoc. w/ underproduction of RBCs. Has an immune basis. Cytokines are released (IL-6), causes and increased uptake and retention of iron in macrophages. This diverts the body's ability to absorb iron into circulation. This limits the amount of iron that's available for RBC production.	Due to autoimmune activity by autoreactive T lymphocytes. Cytotoxic T cells target and destroy the patient's own hematopoietic stem cells. Can also be caused from toxic injury to bone marrow or stem cell defect.	Sudden reduction in total blood volume, and acute loss with increased plasma volume. RBCs available to carry oxygen are decreased.	Chronic loss can be due to depleted iron stores.
Clinical Manifestations	Fatigue, pallor, SOB, tachycardia, irritability, and chest pain.	Fatigue, dyspnea, cardiovascular and cerebral responses, susceptible to infection, at risk of nose bleeds, petechiae, brusing.	Postural hypotension, pain, tissue distention, nerve compression, pain may be localized or deferred. May have numbness and pain in lower extremity from nerve compression.	Pallor, palpitations, dizziness, jaundice, orthostatic hypotension, PICA cravings.
Diagnostic Studies	High serum ferritin, increased iron stores distinguished from iron-deficient anemia, normal folate and cobalamin blood levels.	Hgb, WBC, platelet values are all decreased. Low reticulocyte, serum iron and TIBC may be high. Bone marrow biopsy, aspiration, pathologic exam,	Labs may be normal or high for 2-3 days. RBC mass can become less concentrated. RBCs, Hgb, and Hct may become low.	Stool occult test, endoscopy, colonoscopy, bone marrow biopsy, CBC, blood smear.

Drug Therapy	Best treatment is to correct underlying problem, blood transfusions may be needed if anemia is severe enough. EPO therapy needed if anemia is caused by a renal disease. CBC.	HSCT, immunosuppressive therapy with anti thymocyte globulin (ATG), oral thrombopoietin receptor agonist, high-dose cyclophosphamide, alemtuzumab, androgens.	Replace blood volume, promote coagulation find and stop source of bleed. Blood transfusions, IV fluids to maintain BP. Can take iron supplements, parenteral iron.	Pt. may need to take iron supplements.
Nursing Management	Educate pt. on need for possible blood transfusions, and education on rule out process to correlate anemia with chronic illness or underlying process.	Interventions aimed at injury prevention and education on how to prevent future bleeding and infection.	Educate pt. on interventions needed to help promote the best outcome of blood loss. Educate pt. that they should not need long-term treatment for this type of anemia.	Educate pt. on management of chronic blood loss and processes that involve identifying the source of bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Condition caused by destruction of RBCs at a rate that exceeds production. Macrophages in spleen, liver, and bone marrow destroy RBCs that are old, defective, or moderately damaged.	Iron overload disorder characterized by increased intestinal iron absorption mostly caused by a genetic defect.	Production and presence of increased numbers of RBCs. Can be so great that blood circulation is impaired because of increased blood viscosity and volume.
Clinical Manifestations	Jaundice, enlarged spleen and liver.	Early symptoms are nonspecific, and do not usually develop until after 40 yrs. Of age in men, and 50 yrs. Of age in women. Fatigue, arthralgia, impotence, abdominal pain, and weight loss. Can lead to liver enlargement and cirrhosis, skin pigment changes.	Headache, vertigo, dizziness, tinnitus, visual changes, generalized itching, paresthesia, erythromelalgia, angina, HF, intermittent claudication, thrombophlebitis.
Diagnostic Studies	BUN and creatinine due to build up of Hgb molecules in the renal tubules.	Genetic testing, lab values show high serum iron, TIBC, and high serum ferritin. MRI to measure	High Hgb, hematocrit, RBC mass, bone marrow exam, high WBCs, high platelet count or platelet dysfunction, high

		liver and cardiac iron, liver biopsy.	leukocyte, high uric acid, and high cobalamin levels.
Drug Therapy	Emergency therapy needed w/ aggressive hydration, electrolyte replacement to reduce injury to kidney, corticosteroids, blood products, or removal of spleen. May need folate replacement, immunosuppressive agent, glucocorticoids, and plasma exchange.	Remove excess iron from the body and minimize symptoms. Iron removal of 500mL of blood a week. Iron chelating drugs, deferoxamine given IV or subQ.	Reduce blood volume, viscosity, bone marrow activity. Phlebtomy and low dose ASA to prevent clotting. Myelosuppressive agents.
Nursing Management	Supportive care until causative agent can be eliminated.	Educate pt. on diet changes and to avoid vitamin C, iron supplements, uncooked seafood, and iron-rich foods. Pro	Assist or perform phlebotomy, assess I&Os during hydration therapy, and give myelosuppressive agents. Assess nutrition status.

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.