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	Inadequate diet intake, malabsorption, blood loss, or hemolysis. May occur after certain types of GI surgeries and in malabsorption syndromes. GI blood loss is from peptic ulcers, gastritis, esophagitis, diverticula, hemorrhoids, and cancer. GU blood loss is from menstrual bleeding.	A group of diseases that involve inadequate production of normal Hgb which leads to decreased RBC production. Commonly diagnosed in patients near the Mediterranean Sea and equatorial or near-equatorial regions.	Pernicious anemia Cobalamin is not absorbed because of a lack of intrinsic factor (gastric mucosa) Occurs in patients who had GI surgery or a small bowel resection involving the ileum. Others at risk include Crohn's disease, ileitis, celiac disease, diverticula of small intestine, or chronic atrophic gastritis. Can have a familial predisposition.	Can cause megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation. Common causes include chronic alcohol use, chronic hemodialysis, diet deficiency, drugs interfering with absorption or use of folic acid, increased requirement (pregnancy), and malabsorption syndromes.
Clinical Manifestations	Early symptoms: None Chronic: anemia symptoms Pallor is the most common finding. Glossitis is the second most common (tongue swelling). Cheilitis (lip swelling), headache, paresthesias, and burning sensation of the tongue may also occur	Minor forms are asymptomatic with mild to moderate anemia with microcytosis and hypochromia (small and pale cells), mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Major forms are life-threatening and can cause growth and development deficits by age 2. In adults, symptoms of anemia, jaundice,	Anemia r/t cobalamin deficiency. GI symptoms include anorexia, nausea, vomiting, sore/beefy/red/shiny tongue, abdominal pain. Neuromuscular symptoms include weakness, paresthesia of feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, impaired cognition.	Similar symptoms to cobalamin deficiency. Symptoms may be attributed to coexisting problems. Thiamine deficiency is often also present with folate deficiency.

		pronounced splenomegaly, hepatomegaly, and cardiomyopathy		
Diagnostic Studies	Decreased RBC, HCT, serum iron, ferritin, folate, or vitamin B12. Increased/decreased reticulocytes, LDH, bilirubin, transferrin. Testing may be done to determine cause of iron deficiency ex stool occult blood test, endoscopy, colonoscopy.	Monitor CBC, liver, heart, and lung function	CBC, red blood cells appear larger and have abnormal shapes. Low serum cobalamin levels.	CBC, serum folate is low with a normal serum cobalamin level
Drug Therapy	Oral preparations or IV iron supplements. Patient may need blood transfusion. Iron PO should be taken one hour before meals. Take with vitamin C or orange juice to enhance absorption, iron is best absorbed in an acidic environment. May be taken with meals to reduce GI effects.	Minor does not need treatment because the body adapts to reduced Hgb. Major treatment includes blood transfusions, oral deferasirox or deferiprone, or IV or subQ deferoxamine.	Cobalamin replacement therapy (parenteral or intranasal). 1000mcg/day of cobalamin IM for 2 weeks, then weekly until Hgb is normal and then monthly for life.	Supplements – 1-5 mg/day PO. Duration of treatment depends on reason for deficiency.
Nursing Management	Treat the underlying problem ex reduced intake, poor iron absorption. Replace iron with supplements or diet changes. Teach patient sources of iron or encourage a supplement if nutrition is adequate. If due to blood loss, patient may require packed RBC transfusion.	Patient education about modes of drug therapy and treatment, monitoring of liver, heart, and lungs. Patient may require a splenectomy. Hematopoietic stem cell transplantation is the only cure but often the risks outweigh the benefits.	Assess for neurologic problems that are not corrected by replacement therapy. Implement measures to reduce the risk for injury from the decreased sensitivity to heat and pain. Protect the patient from falls, burns, and trauma. Patient may need physical therapy is neurologic symptoms are not relieved with treatment.	Teach patient to eat foods high in folic acid (green leafy vegetables, enriched grain products/breakfast cereals, and beans),

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Develops after 1-2 months of disease inactivity. Causes include cancer,	The patient has peripheral pancytopenia	Occurs with sudden bleeding from trauma, surgery	Sources are similar to those of iron deficiency anemia.

	autoimmune and infectious disorders, HF, or chronic inflammation. Bleeding episodes can contribute to anemia of chronic disease.	(decrease in all blood cell types) and hypocellular bone marrow. Most aplastic anemias are r/t autoimmune activity by autoreactive T lymphocytes. Other causes include toxic injury to bone marrow stem cells or an inherited stem cell defect.	complications, and problems that disrupt vascular integrity. Leads to hypovolemic shock. If blood loss is gradual, the body maintains its blood volume by slowly increasing plasma volume. This preserves fluid volume but decreases the number of RBC able to carry O2.	Effects are r/t depleted iron stores. Identify the source and stop the bleeding.
Clinical Manifestations	Underproduction of RBC's and mild shortening of RBC survival. RBCs are usually normocytic, normochromic, and hypoproliferative. Anemia is mild but can become severe if underlying disorder is not treated.	Can manifest abruptly or insidiously over weeks and months. Varies from mild to severe. Symptoms may be caused by suppression of any or all bone marrow elements. General symptoms of anemia. Patient with neutropenia is susceptible to infection. Patient is at increased risk for septic shock and death. Thrombocytopenia can lead to bleeding disorders.	Symptoms are determined by amount of blood loss (in mL) 500: None or rare vasovagal syncope 1000: No detectable s/s at rest, increased HR with exercise and slight postural hypotension 1500: Normal supine BP and HR at rest, postural hypotension and increased heart rate with exercise. 2000: BP, central venous pressure, and cardiac output below normal at rest; air hunger; rapid, thready pulse and cold, clammy skin 2500: Shock, lactic acidosis, potential death	Symptoms are related to the cause of chronic blood loss and anemia. Common chronic causes of blood loss include bleeding ulcer, hemorrhoids, menstrual and postmenstrual blood loss.
Diagnostic Studies	High serum ferritin and increased iron stores distinguish it from iron deficient anemia.	Hgb, WBC, platelet values are decreased. Bone marrow biopsy, aspiration, and pathologic	RBS, Hgb, and hct levels are low and reflect blood loss after 2-3 days (when blood loss is sudden).	Monitor CBC

		examination will be done to confirm laboratory findings. The marrow in aplastic anemia is hypocellular with increased yellow marrow.		
Drug Therapy	Treat underlying cause, may require blood transfusions but not for long term management.	Limit the number of blood transfusions which increase the risk of HSCT graft rejection. Eltrombopag can increase platelet counts, high dose cyclophosphamide, alemtuzumab, or adrogens may be helpful for patients who are not responding to other treatment modes.	Blood transfusions are used depending on the volume lost. If large volume is lost, whole blood, platelets, plasma, and cryoprecipitate may be given because large volumes of RBCs dilute the coagulation system. IV fluids are used in emergency situations. Amount of infusion varies depending on solution used. No need for long term treatment.	Patient may need iron supplementation.
Nursing Management	Treat the underlying problem. If anemia is severe, blood transfusions may be needed but are not recommended for long term treatment.	Prevent infection and bleeding. Nursing interventions r/t pancytopenia.	Replace blood volume to prevent shock, promote coagulation to prevent further bleeding, find the source of the bleeding and stop blood loss.	Identify the source and stop the bleeding. Educate on dietary changes needed to maintain iron.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Results from hemolysis of RBCs from extrinsic factors: physical destruction, antibody reactions, infectious agents and toxins.	An iron overload disorder characterized by increased intestinal iron absorption. Genetic defect is the most common cause.	The production and presence of increased number of RBCs. Blood circulation becomes impaired as a result. There is primary (increased WBC, RBC, platelets leading to increased blood viscosity and volume and congestion of organs and tissues with blood) and secondary (hypoxia driven or hypoxia

			independent) polycythemia
Clinical Manifestations	Symptoms depend on the causative agent.	Symptoms do not develop until after age 40 in men and after age 50 in women. Early symptoms are nonspecific including fatigue, arthralgia, impotence, abdominal pain, and weight loss. Later symptoms include enlarged liver, pancreas, heart, joints, and endocrine glands leading to diabetes, skin bronzing, cardiac issues, arthritis, and testicular atrophy.	Circulatory manifestations result from hypertension due to hypervolemia and hyperviscosity (headache, vertigo, dizziness, tinnitus, and visual changes). Generalized itching from a hot bath can be a striking symptom. The patient may have angina, HF, intermittent claudication, and thrombophlebitis, the manifestations of which result from blood vessel distension, impaired blood flow, circulatory stasis, thrombosis, and tissue hypoxia r/t hypervolemia and viscosity.
Diagnostic Studies	CBC, Hgb/Hct is decreased	Laboratory values show high serum iron, TIBC, and serum ferritin. Testing for genetic mutations confirms diagnosis. MRI can measure liver and cardiac iron. Liver biopsy quantifies the amount of iron and established the degree of organ damage.	High Hgb, Hct, and RBC mass; bone marrow examination showing hypercellularity of RBC, WBC, and platelets; presence of genetic mutations
Drug Therapy	Folate replacement, immunosuppressive agents may be used to prevent RBC destruction.	Iron-chelating drugs may be used. Iron removal can also be achieved by removing 500mL of blood weekly until iron stores are depleted. Then, the blood is removed less frequently in order to maintain normal levels of iron within the body.	Treatment is to reduce blood volume and viscosity and bone marrow activity. Phlebotomy is the main treatment to reduce Hct to keep it <45%. Myelosuppressive agents, including hydroxyurea, or busulfan may be given.
Nursing Management	General supportive care until the causative agent can be eliminated or made less injurious to RBCs. Be ready to administer emergency therapy including aggressive rehydration and electrolyte replacement to reduce the risk of kidney injury caused by Hgb clogging the kidney tubules and subsequent shock	Educate the patient to avoid vitamin C and iron supplements, uncooked seafood, and iron-rich foods. Diabetes, and heart failure management and education. Education on treatment modes and management of other symptoms including liver failure, cirrhosis, and liver cancer.	Assess intake and output during hydration therapy to avoid fluid overload or fluid deficit. Educate about side effects of drugs. Assess patient's nutrition status. Begin activities and drug therapy to prevent 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.