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	May develop from inadequate diet intake, malabsorption, blood loss, or hemolysis.	A group of diseases involving inadequate production of normal RBC, which decreases RBC production. Due to an absent or reduced globulin protein. -Autosomal recessive genetic basis.	-Most common cause is deficiency is pernicious anemia. Caused by an absence of intrinsic factor. Known as a disease of insidious onset, begins in middle age or later after 40 generally, 60 being the most common age of onset, occurring most often in blacks and northern European descent. Results in the loss of IF secretion of gastric mucosal cells or impaired absorption of cobalamin in the distal ileum.	Can be caused by chronic alcohol use, chronic hemodialysis, diet deficiency, drugs that interfere with absorption or use of folic acid, pregnancy, celiac disease, crohns disease, small bowel resection.
Clinical Manifestations	Early course may have no symptoms. As it advances the pt might have pallor, glossitis (inflammation of the tongue), cheilitis (inflammation of the lips). May report headache, paresthesia, and a burning sensation of the tongue.	-Thalassemia minor often is asymptomatic. Mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. -Pale, jaundice, pronounced splenomegaly, hepatomegaly and cardiomyopathy, chronic bone	Usually develops due to tissue hypoxia. GI manifestations including sore, red, beefy, and shiny tongue. Anorexia, N/V, abdominal pain, Neuromuscular manifestations such as weakness, impaired cognition. May take years to develop.	Develops insidiously and may be attributed to other coexisting problems. GI problems include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Thiamine deficiency may cause neurologic symptoms.

		marrow hyperplasia, expansion of bone marrow. Diabetes, growth retardation, hypogonadism.		
Diagnostic Studies	Stool occult blood test to determine the cause. Endoscopy and colonoscopy to detect GI bleeding, Bone marrow biopsy if other tests are inconclusive.	Hgb/HCT MCV Reticulocytes Serum Iron TIBC Transferrin Ferritin Bilirubin Serum B12 Folate	Hgb/HCT MCV Reticulocytes Serum Iron TIBC Transferrin Ferritin Bilirubin Serum B12 Folate	Hgb/HCT MCV Reticulocytes Serum Iron TIBC Transferrin Ferritin Bilirubin Serum B12 Folate
Drug Therapy	Enteric coated or sustained release capsules – counterproductive and expensive. Take one hour before meals and with vitamin C for best absorption. -Ferrous sulfate. -IV iron dextran.	Oral deferasirox, Exjade, JadeNu, deferiprone, (ferriprox), or IV or subQ deferoxamine (desferal) new therapy subQ every 21 days luspatercept-aamt (revlozyl)	Parental B12 or intranasal cyanocobalamin (nasocobal). IM cobalamin daily for 2 weeks then weekly until Hgb is normal then monthly for life.	Replacement therapy, 1 to 5 mg PO daily,
Nursing Management	-Treat the underlying problem, reduce intake of alcohol, or reason for poor iron absorption. Teach about foods high in iron. May need transfusion of packed RBC.	-Blood transfusions or exchange of transfusion in conjunction with chelating agents that bind to iron. Monitor heart, liver, and lung function. Provide treatment as needed.	Assess for neuro problems that are not corrected with therapy. Implement measures to reduce the risk of injury from the decreased sensory to pain and heat. Protect patient from heat, falls, and trauma.	Teach to eat foods that are 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	Also called anemia of inflammation, develops 1 to 2 months after disease activity. Causes include cancer, autoimmune and infectious disorders (HIV) HF, or chronic inflammation.	Patients have peripheral blood pancytopenia and hypocellular bone marrow. Due to autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patients' own hematopoietic stem cells.	Sudden reduction in the total blood volume can lead to hypovolemic shock from surgeries, trauma and problems that disrupt vascular integrity.	Acute loss that is more gradual, the body maintains its blood volume by slowly increasing the plasma volume.

Clinical Manifestations	Bleeding episodes that can develop over time. Nosebleeds, petechiae, bruising. Cardiovascular and cerebral symptoms.	Can manifest abruptly or insidiously. Fatigue, dyspnea, Cardiovascular and cerebral responses. Neutropenia, risk for shock and death. Thrombocytopenia can lead to bleeding such as petechiae, bruising, nosebleeds.	Manifestions present as the bodies response to try and maintain an adequate blood volume. Pt may have pain. Or pain and numbness. Focus more on the patients S/S rather than diagnostic studies as these won't reflect blood loss right away. Hypotension.	Pain, numbness, dyspnea, cardiac dysrhythmias, hypotension, Clammy skin, thready pulse and cold.
Diagnostic Studies	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood.	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood.	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood.	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood.
Drug Therapy	-EPO therapy, -Treat the underlying cause.	May receive immunosuppressive therapy with anti-thymocyte globulin ATG and cyclosporine, ORAL eltrombopaq can increase platelet count. Should also receive an iron bonding agent to prevent iron overload.	Replace blood volume deficit and possibly the use of an iron supplement. IV fluid replacement therapy.	Replace blood loss, packed RBC, whole blood, platelets, plasma, cryoprecipitate, IV fluid replacement therapy.
Nursing Management	Correct underlying cause. Administer medications as ordered. Monitor and document S/S. Gather Health Hx.	Identifying and removing the causative agent and providing supportive care until the pancytopenia resolves. Preventative measures to prevent infection and bleeding.	Assess for pain or absence of pain, find the source of bleeding, and correct it, carefully monitor blood loss/drainage.	Assess for pain or absence of pain, Find, and correct the source of bleeding, carefully monitor blood loss/drainage

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
	Results from the hemolysis of RBC from extrinsic	An iron overload disorder characterized by increased	Production and presence of increased numbers of RBC

Etiology	factors. These factors include physical destruction, antibody reactions, and infectious agents and toxins.	intestinal iron absorption. Genetic defect is the most common cause. May occur with diseases such as sideroblastic anemia and liver diseases.	can be primary or secondary. Primary is a chronic myeloproliferative disorder that includes an increased number of RBC, WBC, platelets and leads to an enhanced blood viscosity and blood volume and congestion of organs and tissues with blood. Secondary can be either hypoxia driven or hypoxia independent. Hypoxia drives the kidneys to make EPO and stimulate RBC production. Hypoxia independent is caused by cancer or a tumor that produces EPO. Splenomegaly does not happen with secondary.
Clinical Manifestations	Tachypnea, dyspnea, headache, increased HR, pale skin and mucous membranes, decreased RBC, hypotension	Fatigue, arthralgia, impotence, abdominal pain, weight loss, liver enlargement and cirrhosis, diabetes, skin pigment changes (bronzing), cardiomyopathy, arthritis, and testicular atrophy.	Splenomegaly, hepatomegaly, Headache, vertigo, dizziness, tinnitus, and visual changes, generalized itching, paresthesia and erythromelalgia (painful burning and redness of the hands and feet) impaired blood flow.
Diagnostic Studies	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood	HX and physical Hgb and HCT RBC count Reticulocyte count Serum iron Serum transferrin TIBC Stool exam for occult blood
Drug Therapy	-Folate replacement, immunosuppressive agents may be used. Such as glucocorticoids or rituximab. For severe cases cyclosporine may be used. Plasma exchange and eculizumab are also options.	Iron chelating drugs may be used. Deferoxamine chelates and removes iron via the kidneys. Deferasirox and deferiprone are oral drugs.	Aspirin to prevent clotting. Fluids to reduce blood viscosity. Myelosuppresive drugs such as hydroxyurea, busulfan. Ruxolitinib,
Nursing Management	Be ready to institute appropriate emergency therapy. Supportive care such as administer	Manage organ involvement such as diabetes. Educating the client to avoid vitamin C and iron supplements,	Reducing blood volume and viscosity and bone marrow activity. Phelobotomy is mainstay of

	glucocorticoids and blood products or removing the spleen.	uncooked seafood, and iron rich foods. Remove excess iron from the body and minimize symptoms the patient may have.	treatment. Removal of blood daily. Hydration therapy to reduce blood viscosity, and aspirin to prevent clotting. Assess I and O, Observe and educate patients about drug side effects assess their nutrition status, GI symptoms and pain. Assess for complications at each encounter.

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.