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 an inadequate diet intake, malabsorption, blood loss, or hemolysis. Blood loss is a major cause. Commonly GI blood loss.	A group of diseases involving an adequate production of normal Hgb which decreases RBC production. Commonly found in those of ethnic origins near the Mediterranean Sea or equatorial regions of southeast Asia, the Middle East, India, Pakistan, China, southern Russia, and Africa	Normally the parietal cells of the gastric mucosa secrete IF, without IF we do not absorb cobalamin. Can occur in pt. who had GI surgery or a small bowel resection.	Can cause megaloblastic anemia, Folic acid is needed for DNA synthesis.
Clinical Manifestations	Early on there may be no symptoms. Pallor is the most common finding, glossitis is the second most common. Pt may also report cheilitis, headache, paresthesia, burning sensation of the tongue.	Often Asymptomatic, they have mild to moderate anemia with microcytosis, mild splenomegaly, bronze skin color, and bone marrow hyperplasia.	GI - Sore, red, beefy, shiny tongue. Anorexia, nausea, vomiting, abdominal pain Neuromuscular – weakness, paresthesia of feet and hands, ataxia, reduced vibratory and position senses, muscle weakness, impaired cognition	Symptoms may be from other coexisting problems. GI – stomatitis ,cheilosis, dysphagia, flatulence, diarrhea, thiamine deficiency, neurologic symptoms
Diagnostic Studies	Stool occult blood test, hgb, hct, RBC, reticulocyte count, serum iron, serum ferritin, serum transferrin, TIBC, B12 folate	hgb, hct, RBC, reticulocyte count, serum iron, serum ferritin, serum transferrin, TIBC, B12 folate	hgb, hct, RBC, reticulocyte count, serum iron, serum ferritin, serum transferrin, TIBC, B12 folate	hgb, hct, RBC, reticulocyte count, serum iron, serum ferritin, serum transferrin, TIBC, B12 folate
Drug Therapy	Oral – ferrous sulfate or ferrous gluconate IM or IV – iron dextran, sodium ferrous gluconate, iron sucrose	Drugs used include oral deferasirox, deferiprone, subcutaneous deferoxamine, luspatercept-aamt	Cobalamin administration, parenteral vitamin b12, intranasal, cyancobalamin. 1000 mcg/day for 2 weeks, weekly until Hgb is normal, then monthly	Treat with replacement therapy, duration depends on reason 1-5 mg/day by mouth
	Treat underlying	Minor does not need	Assess for neurologic	Teach to eat foods

Nursing Management	problem, replace iron, teach about foods with good sources of iron, may need packed RBC transfusion	treatment because the body adapts to the reduction of normal Hgb. Major needs treatment like blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron.	problems that are not corrected by replacement therapy, measures to reduce the risk of injury from the decreased sensitivity to heat and pain, protect from falling burns and trauma, administer meds	high in Folic acid, treat underlying problem, replacement therapy administration

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Develops 1 to 2 months after disease activity, caused by cancer, autoimmune and infectious disorders, HF, chronic inflammation. Underproduction of RBC and mild shortening of RBC survival	Due to autoimmune activity by autoreactive T lymphocytes, cytotoxic T cells target and destroy the pt. own hematopoietic stem cells	Occurs with sudden bleeding from trauma or surgery complications, can lead to hypovolemic shock and decreased O2 to the tissues	Cause destruction of red blood cells at a rate that exceeds production. Can occur with problems intrinsic or extrinsic to the RBC
Clinical Manifestations	Same as iron deficiency anemia	Manifest abruptly or insidiously can vary from mild to severe, fatigue, dyspnea, infection	Pain, internal bleeding, numbness, shock	Anemia, jaundice,
Diagnostic Studies	High serum ferritin and increased iron stores distinguish it from iron deficiency anemia	Hgb, WBC, platelet values are decreased, reticulocyte, serum iron, TIBC, RBC, bone marrow biopsy	Plasma, RBC, Hgb, Hct	RBC Hgb, liver and kidney labs
Drug Therapy	Correct underlying problem, administer blood if necessary	ATG, cyclosporin, eltrombopag, thrombopoietin receptor agonist, cyclophosphamide, alemtuzumab, androgens	IV fluids for volume replacement, iron supplements	Based on symptoms and lab values
Nursing Management	Correct underlying problem, blood transfusions, EPO therapy	Identify and remove causing agent, supportive care, blood transfusion	Replace blood volume to prevent shock, promote coagulaitonto prevent further bleeding, find the source of the bleed and stop it, blood transfusions	Maintain renal function, correct underlying problem, blood transfusions

Table 3	**Acquired Hemolytic**	**Hemochromatosis**	**Polycythemia**

	Anemia		
Etiology	Results from hemolysis of RBC from extrinsic factors such as physical destruction, antibody reactions, and infectious agents and toxins	Increased intestinal iron absorption. A genetic defect is the most common cause. It may occur with diseases such as different types of anemia and liver disease and the chronic blood transfusions used to treat thalassemia and SCD	There is primary and secondary polycythemia. Primary involves increased production not only of RBCs but also WBCs and platelets. And secondary hypoxia stimulates the kidneys to make a PO which stimulates RBC production.
Clinical Manifestations	Toxins to RBC, kidney injury, may need spleen removed	Symptoms usually do not develop until after age 40 and men and after age 50 and women. Early symptoms are nonspecific, fatigue, impotence, abdominal pain, weight loss, arthralgia. Later symptoms include liver enlargement, psoriasis, diabetes, skin pigment changes, heart problems, arthritis, testicular atrophy	Circulatory manifestations, hypertension, hypervolemia, hyperviscosity, headache, vertigo, dizziness, tinnitus, visual changes, itching, burning and redness of the hands and feet, intermittent claudication, impaired blood flow, thrombosis, and tissue hypoxia.
Diagnostic Studies	hgb, hct, RBC, reticulocyte count, serum iron, serum ferritin, serum transferrin, TIBC, B12 folate	Serum iron, TIBC, serum ferritin, MRI, liver biopsy	Hgb, hematocrit, RBC, bone marrow examination, WBC, platelets, presence of JAK2, V617F or JAK2 exon mutation, EPO level, leukocyte alkaline phosphase, uric acid, cobalamin
Drug Therapy	Corticosteroids, blood products, folate replacement, glucocorticoids, immunosuppressant	Iron-chelating drugs, deferoxamine, deferasirox, deferiprone	Myelosuppressive agents, Ruxolitinib
Nursing Management	Be ready to give emergency therapy, prevent kidneys from clogging, supportive care	Teach to avoid vitamin C and iron supplements, uncooked seafood, and iron rich foods. Manage problems from organ involvement, supportive care	Reduce blood volume and viscosity and bone marrow activity, phlebotomy, assess intake and output, observe patient and teach them about drug side effects, assess nutrition status, assess symptoms

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.