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	Develop from inadequate diet intake, malabsorption, blood loss, or hemolysis. May occurs after certain types of GI surgery. May exist for a long time before the problem is found.	Group of disease involving inadequate production of normal hgb which decreases RBC production. Absent or reduced globulin protein.	The absence of intrinsic factor. Without IF we do not absorb cobalamin. Gastric mucosa does not secrete IF because gastric mucosal atrophy or autoimmune destruction of parietal cells. Occurring in those who had GI surgery, or small bowel resection. Patients with chron's, ileitis, celiac disease, diverticula or small intestine.	Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	In early iron deficiency patient may not have any symptoms. As the disease becomes chronic patient may feel lethargic, apathy, or fever. Pallor is a common finding, Glossitis, and cheilitis. May report headache, paresthesia, burning of the tongue.	Thalassemia minor is often asymptomatic, have mild to mod anemia with microcytosis and hypochromia, mild splenomegaly, bronzed skin color, and bone marrow hyperplasia. Pale from anemia, jaundice	GI manifestations include sore, red, beefy, shiny tongue; anorexia, nausea, vomiting, and abdominal pain. Nuero: weakness, paresthesia of hands and feet, reduced vibratory and position senses, ataxia, muscle weakness, impaired cognition. May take several months to years to develop manifestations.	Develops insidiously. Symptoms attributed to chron's disease or esophageal varices. DI problems stomatitis, cheilosis, dysphagia, flatulence, and diarrhea. Can cause neurologic symptoms.
Diagnostic Studies	Low Hgb/hct, low mcv, N or slight (high/low) reticulocytes, low	Low hgb/hct, MCV N or low, recticulocytes high, serum iron high,	Hgb/hct low, mcv high, reticulocytes N or low, serum iron N or high, tibc N,	Hgb/hct low, mcv high, reticulocytes N or low, serum iron N or high, tibc N,

	serum iron, high tibc, N or low transferrin, Low ferritin, N or low billi, N serum b12, N folate. Stool occult blood test, bone marrow biopsy	TIBC low, transferrin low, ferritin N or high, billi high, serum b12 N, folate low	transferrin slightly high, ferritin high, billi N or slightly high, serum b12 low, folate N. Upper GI endoscopy and biopsy of gastric mucosa.	transferrin slight high, ferritin high, billi N or slight high, serum b12 N, folate low.
Drug Therapy	Identify and treat underlying cause. Ferrous sulfate or gluconate. Iron dextran, iron sucrose, Packed RBCs	Minor does not need treatment because the body will adapt, major will need blood transfusion or exchange transfusions in conjunction with chelating agents that bind to iron. Drugs used: deferasirox, deferiprone, IV deferoxamine, new therapy called luspatercept-aamt	Parenteral vitamin b12 or intransal cyanocobalamin. 1000mcg/day cobalamin IM for 2 weeks then weekly until hgb is normal and then monthly for life	Replacement therapy. 1 to 5mg/day by mouth
Nursing Management	Assess hgb and RBC to evaluate response to therapy. Stress adherence to diet and drug therapy. Lifelong therapy patients are at a potential risk for liver problems.	Monitor liver heart, and lung function and provide treatment as needed. Hematopoietic stem cell transplant is the only cure (risks outweigh its benefits)	Assess for neurologic problems. Reduce risk for injury from decreased sensitivity to heat and pain. Protect patient from falling, burns, and trauma. May need physical therapy.	Eat foods high in folic acid (green leafy vegetables, orange juice, peanuts, avocado).

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Chronic disease associated with underproduction of RBCs and mild shortening of RBC survival. This type has an immune basis. The cytokine released causes an increased uptake and retention of iron within macrophages.	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	Occurs with sudden bleeding from trauma, surgery complications, and problems that disrupt vascular integrity.	Similar to those of iron deficiency(bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss)

		inherited stem cell defect.		
Clinical Manifestations	Fatigue, paleness of skin, lightheadedness, SOB, irritability, chest pain	Manifest abruptly or insidiously over weeks to months. Varies from mild to severe. Fatigue and dyspnea as well as cardio and cerebral responses. Patients with neutropenia are at risk for infection, septic shock and death. Thrombocytopenia can lead to bruising.	From acute blood loss are caused by the body's attempt to maintain adequate blood volumes and meet O2 requirements. Assess for pain, numbness, shock.	Similar to those of iron deficiency feel lethargic, apathy, or fever.
Diagnostic Studies	High serum ferritin and increased iron. Normal folate and cobalamin blood levels.	Lab studies confirm diagnosis. Affects all marrow elements, hgb, WBC, platelet values are decreased. Bone marrow biopsy, aspiration, and pathologic examination are done to confirm lab findings.	Sudden blood loss plasma volume has yet has a chance to increase. Lab data does not reflect RBC loss. Values may seem normal or high for 2-3 days. Then RBC, hgb, hct, levels are low and reflect actual blood loss.	Blood test, stool test. Endoscopy to find source of bleed
Drug Therapy	Correct underlying problem. Blood tranfusions may be needed.	Identify and remove causative agent. Supportive care. Blood transfusions, immunosuppressive therapy.	Blood transfusions (packed RBCs). Large amount loss can do whole blood, platelets, plasma, and cryoprecipitate. IV fluids, iron supplements	Iron supplements
Nursing Management	Monitor labs, nuero status	Preventing complications from infection and bleeding.	Monitor drainage tubes and dressings. Should start to correct self once found bleed, control loss, and replace volume and fluid. No long term treatment.	Identifying the source and stopping the bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
	Results from hemolysis of RBCs from extrinsic	Iron overload disorder characterized by increased	Production and presence of increased numbers of

Etiology	factors. Physical destruction, antibody reactions, infectious agents and toxins.	iron absorption. Most common cause is genetic defect causing diseases such as sideroblastic anemia and liver disease.	RBCs. Increased numbers in RBC can be so great that blood circulation is impaired because of increased blood viscosity and volume.
Clinical Manifestations	Weakness, paleness, jaundice, dark colored urine, fever, inability to do physical activity, heart murmur	Symptoms do not develop til after age 40 in men, and 50 in women. Fatigue, arthralgia, impotence, abdominal pain, weight loss. Later enlarged liver and cirrhosis. Can cause diabetes, skin pigment changes, heart problems, arthritis, testicular atrophy.	Fatigue, weakness, dizziness, SOB, visual disturbances, nose bleeds, bleeding gums, bruising, heavy menstrual periods, tinnitus, generalized itching, angina, thrombosis.
Diagnostic Studies	Labs: hgb/hct low, MCV N or high, reticulocytes high, serum iron N or high, TIBC N or low, transferrin N, ferritin N or high, billi high, serum b12 N, Folate N	Lab values show high serum iron, TIBC, and serum ferritin, Testing for known genetic mutations. MRI to measure liver and cardiac iron. Live biopsy.	High hgb, hematocrit, and RBC mass. Bone marrow examination showing hypercellularity of RBCs, WBCs, and platelets. Prescence of JAK2 V617F or JAK2 exon 12 mutation. High WBC count with basophilia and neutrophilia, high platelet count and platelet dysfunction, normal or high leukocyte alkaline phosphatase, uric acid, and cobalamin levels.
Drug Therapy	Aggressive hydration and electrolyte replacement to reduce the risk of kidney injury. Corticosteroids and blood products or removing spleen. Folate replacement, immunosuppressive agents.	Removal of iron 500ml of blood each week until iron stores are depleted. Iron-chelating drugs. Deferoxamine chelates, deferasirox and deferiprone. Diet changes avoiding vitamin C and iron supplements, uncooked seafood, and iron rich food.	Treatment aims to reduce blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment reduce hematocrit to 45%. Can become iron deficient. Hydration therapy, low dose aspirin.
Nursing Management	Supportive care	Manage problems from organ involvement with usual treatment of the problem.	Assist with or perform phlebotomy, assess intake and output during hydration therapy to avoid fluid overload or deficit. Assess nutrient 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.