Unit 7: Hematology
Chapter 29 & 30
ONLINE CONTENT (1.5 H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 20, 2023 at 0800. Please be sure to bring a copy to class on March 20, 2023.

Table 1	**Iron Deficiency Anemia**	**Thalassemia**	**Cobalamin (Vitamin B_{12}) Deficiency**	**Folic Acid Deficiency**
Etiology	- May develop because of inadequate dietary intake, malabsorption, blood loss, or hemolysis - Iron malabsorption may occur after certain types of GI surgery and in malabsorption syndromes - Iron absorption occurs in the duodenum - Blood loss occurs from the GI and GU systems, which is a major cause of iron deficiency in adults	- Group of diseases involving adequate production of normal hemoglobin, which decreases normal RBC production - Due to an absent or reduced globulin protein - A-Globin chains are absent or reduced in a-thalassemia - B-Globin chains are absent or reduced in b-thalassemia	- The parietal cells of the gastric mucosa secrete a protein IF, which is required for cobalamin - Cobalamin is absorbed in the distal ileum - Without IF, cobalamin will not be absorbed - In percutaneous anemia, the gastric mucosa is not secreting IF because of either gastric mucosal atrophy or autoimmune destruction of parietal cells - Can occur in patients who have GI surgery, a small bowel resection involving the ileum, and patients with Crohn's disease, ileitis, celiac	- Causes megaloblastic anemia - Needed for DNA synthesis leading to RBC formation and maturation - Common causes include chronic alcoholism, chronic hemodialysis, dietary deficiency (leafy green vegetables, citrus fruits), drugs interfering with absorption or use of folic acid (methotrexate and antiseizure drugs including phenobarbital and phenytoin), increased requirement (pregnancy), malabsorption syndromes including celiac disease,

			disease, diverticula of the small intestine, or chronic atrophic gastritis. In these cases, cobalamin deficiency results from the loss of IF-secreting gastric mucosal cells or impaired absorption in the distal ileum - Can occur with excess alcohol or hot tea ingestion, smoking, long-term user's pf Hs-histamine receptor blockers and proton pump inhibitors, and those who are strict vegetarians	Crohn's disease, and small bowel resection
Clinical Manifestations	- May not have any symptoms initially - General Manifestations: pallor, jaundice, pruiritis, tachycardia, angina, HF, MI, blurred vision, fatigue, dyspnea, headache, tachypnea, irritability, depression,	- Often asymptomatic when minor - Mild to moderate anemia with microcytosis and hypochromia, mild splenomegaly, bronzed color of the skin, and bone marrow hyperplasia - Someone with thalassemia major is pale	- General manifestations of anemia - GI manifestations: sore, red, beefy and shiny tongue; anorexia; nausea and vomiting - Neuromuscular manifestations: weakness, paresthesia of the feet and the hands, reduced	- Develops insidiously - Symptoms may be attributed to other coexisting problems (cirrhosis, esophageal, varices) - GI problems may include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea - Thiamine

	cheilitis, paresthesia, and burning sensation of tongue	and will display other general symptoms of anemia - Jaundice - Hepatomegaly and cardiomyopathy	vibratory and position senses, ataxia, muscle weakness, and impaired thought processes ranging from confusion to dementia - Cobalamin-deficiency anemia has an insidious onset so it may take several months for manifestations to develop	deficiency, which is often present with folate deficiency, can cause neurologic symptoms
Diagnostic Studies	- Hgb/Hct - MCV - Reticulocytes - Serum Iron - TIBC - Transferrin - Ferritin - Bilirubin - Serum B12 - Folate - Stool occult blood test - Endoscopy - Colonoscopy - Bone marrow biopsy	- Labs including Hgb/Hct, MCV, reticulocytes, serum iron, TIBC, transferrin, ferritin, bilirubin, serum B12, and folate	- The RBCs appear large and have abnormal shapes; this structure contributes to RBC destruction because the cell membrane in fragile; serum cobalamin levels are low - Serum folate levels are reviewed; if they are normal and cobalamin levels are low, it suggests that megaloblastic anemia is due to a cobalamin deficiency - A serum test	- Labs: low Hct/Hgb, high MCV, normal or low reticulocytes, normal or high serum iron, normal TIBC, slightly high transferrin, high ferritin, normal or slightly high bilirubin, normal serum B12, low folate - The serum folate level is low (normal is 5 to 25 ng/mL), with a normal serum cobalamin level

			for anti-IF antibodies may be done that is specific for pernicious anemia - Testing of serum methylmaloni c acid (high in cobalamin deficiency) and serum homocysteine (high in both cobalamin and folic acid deficiencies) helps determine the cause of this type of anemia	
Drug Therapy	- Oral iron; daily dose 150-200 mg in 3 or 4 daily doses - Ferrous sulfate - Ferrous gluconate - Iron dextran - Iron sucrose	- Thalassemia major is managed with blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron - Oral deferasirox or deferiprone - IV or SQ deferoxamine - Ascorbic acid supplements - Folic acid - Zinc supplements	- Parenteral vitamin B12 (cyanocobala min, hydroxocobal amin) or intranasal cyanocobala min is needed - 1000 mcg/day of cobalamin IM for 2 weeks and then weekly until the hemoglobin is normal and then monthly for life - High-dose oral cobalamin and sublingual cobalamin are options for those in whom GI	- Treat with replacement therapy - The usual dosage is 1 mg/day by mouth - The patient with malabsorption or chronic alcoholism may need up to 5 mg/day - The duration of treatment depends on the reason for the deficiency

			absorption is intact	
Nursing Management	- Treat underlying problem such as malnutrition or alcoholism - Replace iron - Increase iron intake by dietary means or supplements - If the deficiency is caused from acute blood loss, a transfusion of packed RBCs may be needed	- Hepatic, heart, and lung function are monitored and treated as needed - Stress the importance of frequent rest periods - Education	- Assess for neurologic difficulties that are not fully corrected by replacement therapy - Implement measures to reduce the risk for injury from the decreased sensitivity to heat and pain related to neurologic impairment - Protect the patient from falling, burns, and trauma - In some people, neuromuscular complications may not be reversible and physical therapy may be needed.	- Teach the patient to eat foods high in folic acid (legumes, asparagus, eggs, leafy greens, citrus fruits)

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	- Can be caused by cancer, autoimmune and infectious disorders (human immunodeficiency virus, hepatitis, malaria),	- About 70% of aplastic anemias are due to autoimmune activity by autoreactive T lymphocytes - The cytotoxic T cells target and destroy the patient's own	- Occurs due to sudden hemorrhage - Causes include trauma, complications of surgery, and conditions or diseases	- Associated with an underproduction of RBCs and mild shortening of RBC survival - The RBCs are usually normocytic, normochromic, and hyperproliferative

	HF, or chronic information - Bleeding episodes can contribute to this disease - Associated with an underproduction of RBCs and mild shortening of RBC survival - The RBCs are usually normocytic, normochromic, and hypo proliferative - The anemia is usually mild - It can become more severe if the underlying disorder is not treated	hematopoietic stem cells - Other anemias may be acquired from toxic injury to bone marrow stem cells or result from an inherited stem cell deficit	that disrupt vascular integrity - A sudden reduction in the total blood volume can lead to hypovolemic shock - If the acute loss is more gradual, the body maintains its blood volume by slowly increasing the plasma volume; this preserves circulating fluid volume, but the number of RBCs available to carry O2 is significantly decreased	
Clinical Manifestations	- Fatigue - Paleness - Lightheadedness - Shortness of breath - A fast heartbeat - Irritability - Chest pain	- Can manifest abruptly over days or insidiously over weeks to months - It can vary from mild to very severe - The patient may have symptoms	- Caused by the body's attempts to maintain an adequate blood volume and meet O2 requirements - Signs and symptoms	- Hypotension - Dizziness - Tired - Shortness of breath - Pale - Fatigue - Weakness - Chest pain

		caused by suppression of any or all bone marrow elements - Fatigue and dyspnea, along with cardiovascular and cerebral responses - The patient with neutropenia is susceptible to infection and at risk for septic shock and death - Thrombocytopenia is manifested by a predisposition to bleeding	the patient has are more important than the laboratory values - Assess the patient's expression of pain - Internal hemorrhage may cause pain because of tissue distention, organ displacement, and nerve compression - With retroperineal bleeding, the patient may not have any abdominal pain; instead, the patient may have numbness and pain in a lower extremity from compression of the lateral cutaneous nerve - The major complication of acute blood loss is shock	
Diagnostic Studies	- CBC - Hgb/Hct - MCV	- Laboratory studies confirm the	- When blood volume	- Laboratory studies - CBC - Hgb/Hct

	- Reticulocytes - Serum Iron - TIBC - Transferrin - Ferritin - Bilirubin - Serum B12 - Folate	diagnosis - Hemoglobin, WBC, and platelet values are decreased - Low reticulocyte count - Assessing various iron studies; serum iron and total iron-binding capacity may be high as initial signs of erythropoiesis suppression - Bone marrow biopsy, aspiration, and pathologic examination may be done	loss is sudden, plasma volume has not yet had a chance to increase - The loss of RBCs is not reflected in lab data and values may seem normal or high for 2 to 3 days - Once the plasma volume is replaced, the RBC mass is less concentrated - RBC, hemoglobin, and hematocrit levels are then low and reflect the actual blood loss	- MCV - Reticulocytes - Serum Iron - TIBC - Transferrin - Ferritin - Bilirubin - Serum B12 - Folate
Drug Therapy	- Ferrous sulfate is the mainstay treatment - Supplementation with iron, folic acid, and vitamin B12	- Treatment for choice for adults younger than 55 years of age who do not respond to the immunosuppressive therapy and who have a human leukocyte antigen-matched, half-matched, or unrelated donor, is an HSCT - For older	- IV fluids used in emergencies include dextran, hetastarch, albumin, and crystalloid electrolyte solutions like LR - The amount of infusion varies with the solution used - Blood	- Supplemental iron may be needed

		adults without a donor, the treatment of choice is immunosuppression with ATG and cyclosporine, steroids, and eltrombopag - Patients who need ongoing supportive blood transfusion should be on an iron-binding agent to prevent iron overload - HSCT and immunosuppressive therapy with antithymocyte globulin, steroids, and cyclosporine or cyclophosphamide have improved outcomes	transfusions like PRBCs can be used depending on the volume lost - If a large volume is lost, platelets, plasma, and cryoprecipitate may be needed because large volumes of RBCs dilutes the patient's own coagulation system - May need supplemental iron - When anemia exists after acute blood loss, oral or parenteral iron preparations will be given	
Nursing Management	- Assist the client in developing a schedule for daily activity and rest; stress the importance of frequent rest periods	- Based on identifying and removing the causative agent when possible and providing supportive care until the pancytopenia reverses - Nursing actions are directed at	- Replace blood volume to prevent shock and finding the source of the hemorrhage and stopping the blood loss - May need	- Assist the client in developing a schedule for daily activity and rest; stress the importance of frequent rest periods - Educate energy-conversation techniques - Instruct the client about medications that may stimulate

	- Educate energy-conversation techniques - Instruct the client about medications that may stimulate RBC production in the bone marrow - Provide supplemental oxygen therapy as needed - Anticipate the need for the transfusion of packed RBCs	preventing complications from infection and hemorrhage	to give blood products - Postoperative monitor the blood loss from various drainage tubes and dressings and implement appropriate actions	RBC production in the bone marrow - Provide supplemental oxygen therapy as needed - Anticipate the need for the transfusion of packed RBCs

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	- Caused by the destruction or hemolysis of RBCs at a rate that exceeds production - The RBCs are normal, but external factors are causing damage - Macrophages in the spleen or liver and bone marrow, destroy RBCs that are	- Caused by a faulty gene that can be passed on to a child by their parents - Linked to a fault in gene called HFE, which effects your ability to absorb iron from food	- The production and presence of increased numbers of RBCs - The increase in RBCs can be so great that blood circulation is impaired because of the increased blood viscosity - Primary: involves RBCs, WBCs, and platelets, with an increased production of all

	old		these blood cells; leads to enhanced blood viscosity and blood volume and congestion of organs and tissues with blood - Secondary: hypoxia driven or hypoxia independent
Clinical Manifestations	- General manifestations of anemia and specific manifestations related to this type of anemia - Jaundice is likely because the increased destruction of RBCs causes an elevation in bilirubin levels - The spleen and liver may enlarge because of their hyperactivity, which is related to macrophage phagocytosis of the defective RBCs	- Symptoms usually do not begin until after 40 years in men and 50 years in women - Early symptoms are non-specific and include fatigue, arthralgia, impotence, abdominal pain, and weight loss - Later, the excess iron accumulates in the liver and causes liver enlargement and eventually cirrhosis -	- Circulatory manifestations are often the first symptoms and include headache, vertigo, dizziness, tinnitus, and visual changes - Generalized pruritis may be a striking symptom and is related to histamine release from an increased number of basophils - Paresthesia erythromelalgia may be present - Angina, HF, intermittent claudication, and thrombophlebitis, which may be complicated by embolization - Petechiae, bruising, nosebleeds, or GI bleeding
Diagnostic Studies	- CBC - Other laboratory tests - Lactate dehydrogenase - Unconjugated bilirubin - Haptoglobin tests - Reticulocyte count	- Blood tests including ferritin and transferrin saturation - MRI - Liver biopsy	- High hemoglobin and RBC count with microcytosis - Low to normal EPO level - High WBC count with basophilia and neutrophilia - High platelet count and platelet dysfunction - High leukocyte alkaline phosphatase, uric acid, and

			cobalamin levels - High histamine levels - Bone marrow examination in polycythemia vera shows hypercellularity of RBCs, WBCs, and platelets
Drug Therapy	- Glucocorticoids, such as prednisone, are usually the first line of treatment - Steroids to stop your immune system from attacking your red blood cells - Rituximab may make steroids work even better	- Deferoxamine; only effective way to remove iron from patients who have been overloaded with iron because of multiple transfusions	- Myelosuppressive agents, such as hydroxyurea, busulfan, and chlorambucil - Ruxolitinib inhibits the expression of the JAK2 mutation, and is given to patients who have not responded to hydroxyurea - Anagrelide can reduce the platelet count and inhibit platelet aggregation low-dose aspirin is used to prevent clotting - A-interferon and pegylated interferon are given to women of childbearing age or those with intractable pruritis - Allopurinol may reduce the number of acute gouty attacks
Nursing Management	- Transfusion of blood - IV fluids - Monitor intake and output - Daily weights	- Avoiding vitamin C supplements, raw selfish, and high alcohol use - Assess fluid intake and output - Phlebotomy	- Assist with or perform the phlebotomy - Assess fluid intake and output during hydration therapy to avoid fluid overload or fluid deficit - If myelosuppressive drugs are used, give the drugs as

			ordered, observe the patient, and teach the patient about the side effects of the drugs - Assess the patient's nutritional status since inadequate food intake can result from GI symptoms of fullness, pain, and dyspepsia - Begin activities and/or medications to decrease thrombus formation - Start active or passive leg exercises and ambulation when possible - Assess patient for complications