Katelyn Howard
Anemia online assignment Unit 7

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, hemolysis. May be inadequate for people with higher iron needs (pregnant, menstruating)	Group of diseases involving inadequate production of normal Hgb, which decreases RBC production. Due to an absent or reduced globulin protein. Commonly found in members of ethnic groups (Mediterranean sea, Asia, middle east, India, Pakistan, china, southern Russia, Africa)	Chronic alcoholism, dietary deficiency, deficiency of gastric intrinsic factors: celiac disease, gastrectomy, gastric bypass, helicobacter pylori, pernicious anemia, increased requirement (pregnancy), intestinal malabsorption	Chronic alcoholism, chronic hemodialysis, dietary deficiency, drugs interfering with absorption or use of folic acid (methotrexate, antiseizure drugs), increased requirement (pregnancy), malabsorption syndromes (celiac disease, crohn's disease, small bowel resection)
Clinical Manifestations	Early: may have no symptoms. As it becomes chronic, may have tachycardia, blurred vision, difficulty swallowing, pallor (most common), glossitis (inflammation of tongue), bone pain, tachypnea, HA, vertigo, cheilitis (inflammation of lips), paresthesia, burning sensation of the tongue	Minor: often asymptomatic. Mild to moderate: microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed skin, bone marrow hyperplasia. Major: life threatening, pale, displays other general symptoms of anemia	Develop because of tissue hypoxia. GI: sore, red, beefy and shiny tongue, anorexia, N/V, abdominal pain, weakness, paresthesia of feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, impaired thought processes ranging from confusion to dementia. Insidious onset, it may take several months for manifestations to develop	Similar to cobalamin deficiency. Develops insidiously. Symptoms may be attributed to other coexisting problems: cirrhosis, esophageal varices. GI problems: stomatitis, cheilosis, dysphagia, flatulence, diarrhea. Can cause neurological symptoms.
Diagnostic Studies	History/physical exam. Hgb & Hct, RBC, reticulocyte, iron, ferritin, transferrin, TIBC, stool exam for occult blood. Endoscopy and colonoscopy. Bone	Hgb/Hct, MCV, reticulocytes, iron, TBIC, transferrin, ferritin, bilirubin, B12, Folate	Hgb/Hct, MCV, reticulocytes, iron, TIBC, transferrin, ferritin, bilirubin, b12, folate. Large RBCs (macrocytic). Folate levels are reviewed, if they are normal and	Hgb/Hct, MCV, reticulocytes, iron, TIBC, transferrin, ferritin, bilirubin, b12, folate (LOW)

	marrow biopsy may be done if other tests are inconclusive		cobalamin levels are low, it suggests that megalobastic anemia is due to a cobalamin deficiency. MMA and serum homocysteine helps determine the cause	
Drug Therapy	Oral: Ferrous sulfate or ferrous gluconate IM or IV: iron dextran, sodium ferrous gluconate, iron sucrose	Minor: no treatment Major: managed with blood transfusions or exchange transfusions in conjunction with chelating agents that bind to iron. Oral deferasirox, deferiprone, or IV or subcutaneous deferoxamine. Ascorbic acid supplements may be needed during chelation therapy, given along with folic acid. Zinc supplements may need needed.	Parenteral vitamin B12 or intranasal cyanocobalamin is needed. Without cobalamin administration, patient will die in 1-3 years. Typical treatment consists of 1000 mcg/day of cobalamin IM for 2 weeks then weekly until Hgb is normal, then monthly for life.	Replacement therapy. Usual dosage is 1 mg/day by mouth. Malabsorption or chronic alcoholism may need up to 5 mg/day. Duration of treatment depends on the reason for it.
Nursing Management	Treat underlying problem, replace iron, teach which foods are good sources of iron. May need oral or parenteral iron supplements. May need blood transfusion. Lean beef, turkey, pork and chicken, fish, legumes, dark green leafy vegetables, whole grain and enriched bread and cereals, beans.	No specific drug or diet therapies are effective in treating. Give transfusions to keep Hgb level around 10 g/dL.	Correct physiologic status deficits, encourage alternating rest and activity. Monitor cardiorespiratory response to activity. Limit number of and interruptions by visitors. Assist the patient in assigning priority activities. Arrange physical activities. Teach patient and caregivers to recognize S/S of fatigue that required reduction in activity. Teach to notify HCP if S/S of fatigue persist. Meat, eggs, enriched grain products, milk and dairy foods, fish (especially salmon)	Correct physiologic status deficits, encourage alternating rest and activity. Monitor cardiorespiratory response to activity. Limit number of and interruptions by visitors. Assist the patient in assigning priority activities. Arrange physical activities. Teach patient and caregivers to recognize S/S of fatigue that required reduction in activity. Teach to notify HCP if S/S of fatigue persist. Green leafy veggies, enriched grain products and breakfast cereals, OJ, PB and avocado.

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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 (HIV), hepatitis, malaria, HF or chronic inflammation. Bleeding episodes can contribute to it as well. Associated with an underproduction of RBCs	About 70% are due to autoimmune activity by auto reactive T lymphocytes. Cytotoxic T cells target and destroy the patients own hematopoietic stem cells.	Occurs because of sudden hemorrhage from trauma, complications of surgery or conditions/diseases that disrupt vascular integrity. Sudden reduction in the total blood volume can lead to hypovolemic shock. If acute loss is more gradual, the body maintains its blood volume by slowly increasing the plasma volume. Although this preserves circulating fluid volume, the number of RBCs available to carry O2 is significantly decreased	Similar to those of iron deficiency anemia (bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss). Effects of chronic blood loss are usually related to depletion of iron stores and considered an iron deficiency anemia. Loss of RBCs exceeds production of new RBCs
Clinical Manifestations	Fatigue, pallor, lightheadedness, SOB, tachycardia, irritability, CP, low RBCs	Fatigue, dyspnea, cardiovascular and cerebral responses. Patient with neutropenia (low neutrophil count) is susceptible to infection and is at risk for septic shock/death. Thrombocytopenia is manifested by a predisposition to bleeding (petechiae, bruising, nosebleeds)	Caused by the body's attempts to maintain an adequate blood volume and meet O2 requirements. 500mL: none or rare vasovagal syncope. 1000mL: no detectable S/S at rest. Tachy with exercise and slight postural hypotension. 1500mL: Normal supine BP and pulse at rest. Postural hypotension and tachy with exercise. 2000mL: BP, central venous pressure, CO below normal at rest, air hunger, rapid thready pulse, cold, clammy. 2500mL: Shock, lactic acidosis, potential death	Extreme fatigue, weakness, pale skin, CP, fast heartbeat, SOB, HA, dizzy, cold hands/feet, inflammation or soreness of tongue, brittle nails, BP drops.
Diagnostic Studies	High serum ferritin and increased iron stores distinguish it from iron deficiency anemia. Normal folate and	Hgb, WBC and platelet values are decreased. Various iron studies. TIBC may be high as initial	When blood volume loss is sudden, plasma volume has not yet had a chance to increase. The loss of RBCs is	CBC, hgb/hct. May perform bone marrow biopsy. High or low iron level.

	cobalamin blood levels distinguish it from megaloblastic anemia from folate and cobalamin deficiencies	signs of erythropoiesis suppression. Bone marrow biopsy, aspiration, and pathologic examination may be done. Increased yellow marrow (fat content)	not reflected in lab data, and values may seem normal or high for 2-3 days. However, once plasma volume is replaced, the RBC mass is less concentrated. Then RBC, hgb/hct are low and reflect the actual blood loss.	
Drug Therapy	Treat underlying disorder. If severe: blood transfusions may be needed but not recommended long term. Erythropoietin therapy is used for anemia related to renal disease and cancer and its therapies. Its use is limited, though because of the increased risk for thromboembolism and death in some patients.	HSCT and immunosuppressive therapy with ATG, steroids and cyclosporine and cyclophosphamide.	IV fluids used in emergencies: dextran, hetastarch, albumin, crystalloid electrolyte solutions like LR. Amount of infusion varies with the solution used. Blood transfusions (packed RBCs) can be used depending on volume lost. If a large volume of blood is lost, platelets, plasma, and cryoprecipitate may be needed because large volumes of RBCs dilutes the patients own coagulation system.	Fluids, blood transfusion, oxygen and possibly iron to help your body build new RBCs.
Nursing Management	Assess risk factors, decrease fatigue, adequate nutrition, maintenance of adequate tissue perfusion, compliance with meds and free from complications	Based on identifying and removing the causative agent (when possible) and providing supportive care until the pancytopenia reverses. Arrange physical activities. Teach about nutritional needs. Use soft bristled toothbrush. Remove fresh flowers.	Identifying the source and stop the bleeding. Supplemental iron may be needed. Replacing blood volume to prevent shock. Finding the source of hemorrhage and stopping the blood loss. IV fluids used in emergencies.	Identifying source and stop the bleeding. Supplemental iron may be needed.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Physical destruction, antibody reactions, and infectious agents and toxins. Physical destruction of RBCs results from the exertion of extreme forces on the cells, such as when	Iron overload disorder. Genetic defect most common. May occur with sideroblastic anemia. May also be caused by liver disease and the chronic blood transfusions used to treat thalassemia and SCD.	Production and presence of increased number of RBCs. The increased can be so great that blood circulation is impaired because of the increased blood viscosity and blood volume. 2 types:

	they travel past prosthetic heart valves, through partially occluded vessels.		primary polycythemia and secondary polycythemia. Primary involves RBCs and WBCs. Secondary is hypoxia driven or hypoxia independent.
Clinical Manifestations	Weakness, paleness, jaundice, dark colored urine, fever, inability to do physical activity, heart murmur	Symptoms usually do not develop until after age 40 in men and after 50 in women. Early symptoms are nonspecific and include: fatigue, arthralgia, impotence, abdominal pain, weight loss. Later, the excess iron accumulates in the liver and causes liver enlargement and eventually cirrhosis. Excess iron is deposited in the liver, pancreas, heart, joints, and endocrine glands, resulting in diabetes, skin pigment changes (bronzing), heart problems, (cardiomyopathy), arthritis and testicular atrophy. Physical exam reveals an enlarged spleen, liver and pigmentation changes in the skin.	HA, vertigo, dizziness, tinnitus, visual changes. Generalized pruritus (often exacerbated by a hot bath) may be a striking symptom. Paresthesias and erythromelalgia (painful burning and redness of hands/feet). May have angina, HF, intermittent claudication and thrombophlebitis.
Diagnostic Studies	Hgb/Hct, MCV, Reticulocytes, iron, TIBC, ferritin, bilirubin	High serum iron, TIBC and serum ferritin. Testing for known genetic mutations confirms the diagnosis. Liver biopsy can quantify the amount of iron and establish the degree of organ damage.	High Hgb and RBC count with microcytosis, low to normal EPO level (secondary has a high level), high WBC count with basophilia and neutrophilia, high platelet count and platelet dysfunction, high leukocyte alkaline phosphate, uric acid, and cobalamin levels and high histamine levels
Drug Therapy	To suppress the RBC destruction, immunosuppressive agents may be used, such as glucocorticoids or rituximab, which is a monoclonal antibody to b cell CD20. For severe cases associated, thrombocytopenia and AKI, plasma exchange and eculizumab to complement protein	Iron removal is achieved by removing 500mL of blood each week for 2-3 years until the iron stores in the body are depleted. Then blood is removed less often to maintain iron levels WNL. Iron chelating agents may be used. Deferoxamine, can be given IV or subcutaneously. Deferasirox and deferiprone oral agents that chelate iron. Dietary changes; avoiding vitamin C and iron supplements, uncooked seafood and iron rich foods.	Myelosuppresive agents, such as hydroxyurea, busulfan, and chlorambucil may be given. Ruxolitinib, is given to patients who have not responded to hydroxyurea. Other therapies: anagredlide, which can reduce platelet count and inhibit platelet aggregation. Low dose ASA. A-IFN- for childbearing age or have intractable pruritus. Allopurinol may reduce number of acute gouty attacks.

Nursing Management	General supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs. Be ready to institute appropriate emergency therapy- aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by hgb clogging the kidney tubules and subsequent shock. Supportive care may include giving corticosteroids and blood products or removing spleen. May need folate replacement. To suppress RBC destruction, immunosuppressive agents may be used.	Management of organ involvement is the same as conventional treatment for these problems. Most common causes of death are cirrhosis, liver failure, liver cancer, and HF. So early diagnosis and treatment is needed for life expectancy to be normal. Avoid animal fat and sugar, limit Vitamin C, reduce red meat… Monitor blood tests. Give medications as prescribed.	May assist with or perform the phlebotomy. Assess fluid intake and output during hydration therapy to avoid fluid overload, or fluid deficit. If myelosuppressive agents are used, give the drugs as ordered, observe the patient, teach them about side effects. Assess 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. Requires ongoing evaluation. Assess patient for complications.