AMSN 2022

Unit 7: Hematology- Red Blood Cell Disorders (Anemia)

Complete this worksheet and do the following:

-Place into the Unit 7: Hematology dropbox by March 21, 2022 at 0800 (1.5H).

-**Have a copy for class on March 21, 2022.**

Page 1	Iron Deficiency Anemia	Thalassemia	Cobalamin Deficiency	Folic Acid Deficiency
Etiology	Blood loss is a major cause of iron deficiency in adults, such as in GI bleeds or a menstruating female. Iron malabsorption may occur after certain types of gastrointestinal surgery and in malabsorption syndrome. Iron deficiency anemia may develop because of inadequate dietary intake, malabsorption, blood loss, or hemolysis. Iron absorption occurs in the duodenum, so malabsorption may occur after a surgical procedure that involves removal or bypass of the duodenum.	Thalassemia Is a group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production. It is due to an absent or reduced globulin protein. a-Globin chains are absent or reduced in alpha-thalassemia, and b-Globin chains are absent or reduced in b-thalassemia. There are two types: thalassemia minor and major. Thalassemia Is commonly found in members of ethnic groups whose origins are near the Mediterranean Sea and equatorial or near-equatorial regions of Southeastern Asia, the Middle East, India, Pakistan,	The most common cause of cobalamin deficiency is pernicious anemia. It is caused by absence of intrinsic factor (IF). Cobalamin deficiency occurs with excess alcohol or hot tea ingestion, smoking, long-term users of H2 histamine receptor blockers and proton pump inhibitors, and those who are strict vegetarians. Cobalamin deficiency can also occur in patients who have had GI surgery (e.g., gastrectomy, gastric bypass); patients who have had a small bowel resection involving the ileum; and patients with Crohn's disease, ileitis, celiac disease,	Common causes of folic acid deficiency are chronic alcoholism, chronic hemodialysis, dietary deficiency, drugs interfering with absorption or use of folic acid such as methotrexate and antiseizure drugs, increased requirement such as pregnancy, and malabsorption syndromes such as Celiac disease, Crohn's disease, and small bowel resection.

		China, Southern Russia, and Africa. Thalassemia is also an autosomal recessive genetic 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 of cobalamin in the distal ileum.	
Clinical Manifestations	In the early course of iron deficiency anemia, the patient may not have any symptoms. As the disease becomes chronic, any of the general manifestations of anemia may develop. Pallor, glossitis (swelling of the tongue), cheilitis (inflammation of the lips), headache, paresthesia, and burning sensation of the tongue are most common clinical manifestations.	Thalassemia Major is a life-threatening disease in which growth, both physical and mental, is often slowed. Jaundice, splenomegaly, hepatomegaly, cardiomyopathy, chronic bone marrow hyperplasia (can cause thickening of the cranium and maxillary cavity) may occur. They are often pale and display other general symptoms of anemia. The symptoms develop in childhood by 2 years of age and can cause growth and developmental deficits. Thrombocytosis after spleen	General manifestations of anemia related to cobalamin deficiency develop because of tissue hypoxia. GI manifestations include a sore, red, beefy, and shiny tongue; anorexia, nausea, and vomiting; and abdominal pain. Typical neuromuscular manifestations include weakness, paresthesia of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired thought processes ranging from confusion to dementia. Because cobalamin	This disease develops insidiously. Thiamine deficiency, which is often present with folate deficiency, can cause neurological symptoms. The patient's symptoms may be attributed to other coexisting problems (e.g., cirrhosis, esophageal varices). GI problems may include stomatitis, cheilosis, dysphagia, flatulence, and diarrhea.

		dysfunction and/or removal may occur. The patient with thalassemia minor is often asymptomatic. The patient has mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed color of the skin, and bone marrow hyperplasia.	deficiency-related anemia has an insidious onset, it may take several months for manifestations to develop.	
Diagnostic Studies	Low Hgb/Hct, low MCV, normal or slight increased or decreased reticulocytes, decreased serum iron, increased TIBC, normal or decreased transferrin, decreased ferritin, normal or decreased bilirubin, normal serum B12, and normal folate. Stool occult blood test, endoscopy, colonoscopy, and bone marrow biopsy.	Decreased Hgb/Hct, normal or decreased MCV, increased reticulocytes, increased serum iron, decreased TIBC, decreased transferrin, normal or increased ferritin, increased bilirubin, normal serum B12, and decreased folate.	The RBCs appear large and have abnormal shapes. A serum test for anti-IF antibodies may be done that is specific for pernicious anemia. Patients with pernicious anemia may undergo an upper GI endoscopy and biopsy of the gastric mucosa at the time of diagnosis and at appropriate intervals afterward. Decreased Hgb/Hct, increased MCV, normal or decreased reticulocytes, normal or increased serum iron, normal TIBC, slightly increased	Decreased Hgb/Hct, increased MCV, normal or decreased reticulocytes, normal or increased serum iron, normal TIBC, slightly increased transferrin, increased ferritin, normal or slightly increased bilirubin, normal serum B12, and decreased folate.

			transferrin, increased ferritin, normal or slightly increased bilirubin, decreased serum B12, and normal folate. Decreased serum cobalamin. Testing of serum methylmalonic acid (MMA) (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 -include name, dosage, route, and nursing interventions	Oral iron tablet 150 or 200 mg by mouth daily. This can be taken in 3 or 4 daily doses, with each tablet or capsule of the iron preparation containing between 50 and 100 mg of iron. Iron should be taken about an hour before meals. Taking iron with vitamin C enhances iron absorption. If the iron deficiency is from acute blood loss, the patient may need transfusion of packed RBCs.	Oral deferasirox 20 mg/kg is a drug used for thalassemia major. Assess patient for rash, GI pain, and bleeding during therapy. Monitor serum ferritin levels and liver function while on this medication. Thalassemia is also managed with blood transfusions that are given to keep the hemoglobin level around 10 g/dL. Chelating agents reduce the	High-dose oral cobalamin and sublingual cobalamin are options for those in whom GI absorption is intact. A typical treatment schedule consists of 1000 mcg/day of cobalamin IM for two weeks and then weekly until the hemoglobin is normal and then monthly for life. Monitor plasma folic acid, vitamin B12, serum potassium and iron levels,	Folic acid deficiency is treated with replacement therapy. The usual dosage is 10 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. While taking folic acid supplements, monitor plasma folic acid levels, hemoglobin,

	Undiluted liquid iron may stain the patient's teeth so it should be diluted and ingested through a straw. Have the patient stay upright for 30 minutes after taking oral forms. If GI side effects occur, the dose and type of supplement may be adjusted.	iron overloading that occurs with chronic transfusion therapy.	hemoglobin, hematocrit, and reticulocyte count. Assess patient for signs of vitamin B12 deficiency such as pallor, neuropathy, psychosis, red and inflamed tongue) before and periodically during therapy.	hematocrit, and reticulocyte count before and periodically during therapy. Also, assess patients for signs of megaloblastic anemia (fatigue, weakness, dyspnea) before and periodically throughout therapy.
Nursing Management -include patient education	Teach patients iron is best absorbed in an acidic environment and that it should be taken on an empty stomach to avoid binding the iron with food. Teach patients which foods are good sources of iron. Teach patients that taking iron with orange juice enhances iron absorption. Teach patients that undiluted liquid iron may stain their teeth so it should be	Teach patients that ascorbic acid should only be taken with chelation therapy because it increases the absorption of dietary iron and that it is given, along with folic acid, if there is evidence of hemolysis. Zinc supplements may be needed since zinc is reduced with chelation therapy and iron supplements should not be given. The patient may need a splenectomy and	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 neurological impairment. Protect the patient from falling, burns, and trauma. In some people, the neuromuscular complications may not be reversible	Teach patients of foods that are high in folic acid. These include green leafy vegetables, and rich green products and breakfast cereals, orange juice, peanuts, and avocados.

	diluted and ingested through a straw. Teach patients to monitor for GI side effects such as heartburn, constipation, and diarrhea. Teach patients that their stools will become black.	hepatic, heart, and lung function should be monitored and treated as needed.	and physical therapy may be needed. Educate that there is a familial predisposition for pernicious anemia, and evaluate patients who have a positive family history of pernicious anemia for symptoms.	

Page 2	Anemia of Chronic Disease	Aplastic Anemia	Acute Anemia due to blood loss	Chronic Anemia due to blood loss
Etiology	Underproduction of RBCs and mild shortening of RBC survival. The RBCs are usually normocytic, normochromic, and hypoproliferative. The cytokines released cause an increased uptake	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 hematopoietic stem cells. Other anemias	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. Although this	The source of chronic blood loss are similar to those of iron deficiency anemia (e.g., bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss). The effects of chronic

	and retention of iron within macrophages. This leads to a diversion of iron from the circulation into storage sites with subsequent limited iron available for erythropoiesis.	may be acquired from toxic injury to bone marrow stem cells or result from an inherited stem cell defect.	preserves circulating fluid volume, the number of RBCs available to carry O2 is significantly decreased.	blood loss are usually related to the depletion of iron stores and considered an iron-deficiency anemia.
Clinical Manifestations	Must be first recognized and distinguished from anemia of other causes. Palpitations, tachycardia, angina, HF, MI, increased PP, systolic murmurs, headache, vertigo, irritability, depression, impaired thought processes anorexia, hepatomegaly, splenomegaly, pallor, jaundice, pruritis, glossitis, smooth tongue, difficulty swallowing, sore mouth, sensitivity to cold, weight loss, lethargy, bone pain, tachypnea, orthopnea, and dyspnea at rest.	Aplastic anemia can manifest abruptly or insidiously over weeks to months. It can vary from mild to severe. General manifestations of anemia, such as fatigue and dyspnea, as well as cardiovascular and cerebral responses, may be seen. The patient may have symptoms caused by suppression of any or all bone marrow elements. The patient with neutropenia is susceptible to infection and is at risk for septic shock and death. Thrombocytopenia is manifested by a predisposition to bleeding.	The manifestations of anemia from acute blood loss are caused by the body's attempts to maintain an adequate blood volume and meet O2 requirements. For 500 mL, none or rare vasovagal syncope. For 1000 mL, no detectable signs or symptoms at rest, tachycardia with exercise, and slight postural hypotension. For 1500 mL, normal supine blood pressure and pulse at rest, postural hypotension, and tachycardia with exercise. For 2000 mL, BP, central venous pressure, and CO below normal at rest, air hunger, rapid thready pulse, and cold, clammy skin.	Palpitations, tachycardia, increased PP, systolic murmurs, angina, HF, MI, anorexia, hepatomegaly, splenomegaly, difficulty swallowing, sore mouth, sensitivity to cold, weight loss, lethargy, pallor, jaundice, pruritis, glossitis, smooth tongue, bone pain, tachypnea, orthopnea, dyspnea at rest, headache, vertigo, irritability, depression, impaired thought processes.

			For 2500 mL, shock, lactic acidosis, and potential death.	
Diagnostic Studies	Normal cobalamin and folate blood levels distinguish it from megaloblastic anemias from folate and cobalamin deficiencies. High serum ferritin and increased iron stores distinguish it from iron-deficiency anemia.	Laboratory studies confirm the diagnosis. Because aplastic anemia affects all marrow elements, hemoglobin, WBC, and platelet values are decreased. Other RBC indices are generally normal. The condition is therefore classified as a normocytic, normochromic anemia. The reticulocyte count is low. The 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. The marrow in aplastic anemia is hypocellular with increased yellow marrow.	When blood volume loss is sudden, plasma volume has not yet had a chance to increase. The loss of RBCs is not reflected in laboratory data, and values may seem normal or high for 2 to 3 days. However, once the plasma volume is replaced, the RBC mass is less concentrated. Then, RBC, hemoglobin, and hematocrit levels are low and reflect the actual blood loss.	Decreased Hgb/Hct, MCV, normal or increased reticulocytes, decreased serum iron, decreased TIBC, normal transferrin, ferritin, normal or decreased bilirubin, normal B12 and folate.
Drug Therapy	If anemia is severe, blood transfusions may be needed, but	Antithymocyte globulin, 1.5mg/kg IV qd x 7-14 days,	Hetastarch, IV 30-60g may be repeated; not to	Supplemental iron may be needed.

-include name, dosage, route, and nursing interventions	are not recommended for long-term treatment. Erythropoietin therapy is used for anemia related to renal disease and cancer and its therapies. epoetin- ESA, SC 150 units/kg 3 times weekly, IV 600 units/ kg weekly. Monitor for symptoms of anemia. Monitor BP before and during therapy. Inform health care professionals if severe HTN or if BP increases.	infuse over a minimum of 6 hours for the first infusion and over at least 4 hours on subsequent days of therapy. Monitor lymphocyte count, WBC, and platelet counts during therapy. Immunization with attenuated live vaccines is not indicated for patients who have recently received AGT.	exceed 90g. In acute hemorrhagic shock, up to 20 mL/kg/hr may be used. Monitor vital signs, CVP, CO, pulmonary capillary wedge pressure, and urinary output before and frequently throughout therapy. Assess patient for signs of vascular overload during and after administration. If fever, wheezing, flu-like symptoms, urticaria, periorbital edema, or submaxillary and parotid gland enlargement occurs, stop infusion and notify health care professional immediately. Antihistamines, epinephrine, corticosteroids, and airway management may be required to suppress this response.	iron dextran, IM/IV total dose (mL)= 0.0476 x (14.8 - actual Hgb) x lean body weight (kg) + 1mL/per 5 kg lean body weight. Divided up and given in small daily doses until total is reached, not to exceed 100 mg/day. Total dose may be diluted and infused over 4-5hr following a test dose of 10 drops.
Nursing Management -include patient education	May decrease bleeding times. Monitor serum ferritin, transferrin, and iron levels to assess the need for concurrent iron	Management of aplastic anemia is based on identifying and removing the causative agent and providing supportive care	Interprofessional care is initially concerned with replacing blood volume to prevent shock and finding the source of the	Management of chronic blood loss anemia involves identifying the source and stopping the bleeding. Assess bowel function for

	therapy. Erythropoietin therapy may cause an increase in WBCs and platelets. Advise patient to notify health care professional immediately if signs and symptoms of blood clots occur. Discuss ways of preventing self-injury in patients at risk for seizures. Advise patient to inform health care professional of medication prior to treatment or surgery.	until the pancytopenia reverses. Nursing actions are directed at preventing complications from infection and hemorrhage.	hemorrhage and stopping the blood loss. The body needs 2 to 5 days to make more RBCs in response to increased erythropoietin. The patient may need supplemental iron because the availability of iron affects the marrow production of RBCs. When anemia exists after acute blood loss, dietary sources of iron will probably not be enough to maintain iron stores. therefore oral or parenteral iron preparations are given. For the postoperative patient, carefully monitor the blood loss from various drainage tubes and dressings and implement appropriate actions. the nursing care for the patient with anemia resulting from acute blood loss will likely include giving blood products.	constipation or diarrhea. Notify healthcare professional and use of appropriate nursing measures should these occur. Monitor BP and HR frequently following IV administration until stable. Rapid infusion rate may cause hypotension and flushing. Assess patient for signs and symptoms of anaphylaxis. Notify healthcare professional immediately if these occur. Keep epinephrine and resuscitation equipment close by in the event of an anaphylactic reaction. Monitor hemoglobin, hematocrit, and reticulocyte values prior to and every 3 weeks during the first 2 months of therapy and periodically therafter.

Page 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	Results from hemolysis of RBC from extrinsic factors which are physical destruction, antibody reactions, and agents and toxins. Hemolytic toxins that can cause hemolysis include oxidative drugs, arsenic, lead, copper, and bee stings/spider bites. Antibodies may destroy RBCs by the mechanisms involved in antigen-antibody reactions. This can be an idiopathic or from other other autoimmune diseases (SLE, leukemia, lymphoma, procainamide, penicillin, ibuprofen, metformin, and chlorpromazine). Traumatic events causing physical destruction are	Hemochromatosis is an iron overload disorder. Although an autosomal recessive genetic defect is the most common cause, it may occur with diseases such as sideroblastic anemia. The genetic link causes increased intestinal iron absorption and, as a result, increased tissue iron deposition. People with hemochromatosis accumulate iron at an increased rate. It can also be caused by liver disease and the chronic blood transfusion used to treat thalassemia and SCD.	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 (hyperviscosity) and volume (hypervolemia). There are two types: primary and secondary polycythemia. Primary is a chronic myeloproliferative disorder. It involves an increased production of RBCs, WBCs and platelets. This leads to enhanced blood viscosity and blood volume and congestion of organs and tissues with blood. Secondary can be either hypoxia driven or hypoxia independent. The genetic link is characterized by polycythemia vera

	hemodialysis, extracorporeal circulation used in cardiopulmonary bypass, and prosthetic heart valves. Force needed to push blood through abnormal vessels, (those that have been burned, irradiated, or affected by valvular disease like diabetes) can physically damage RBCs. RBCs are are fragmented and destroyed as they try to pass through abnormal arterial or venous circularion. The RBCs are sheared as they try to pass by excess platelet aggregation and/or fibrin polymer formation, such as is seen in TTP and DIC. Infectious agents cause hemolysis in three ways: by releasing hemolytic substances (clostridium perfringens), by invading the RBC and destroying its contents, and by generating an antigen-antibody reaction (mycoplasma pneumoniae).		which is associated with mutations in the Janus kinase-2 (JAK2) gene (provides instructions for making a protein that promotes proliferation of cells, especially blood cells from hematopoietic stem cells). Most cases are acquired during a person's lifetime and are present only in certain cells.
	Clinical manifestations include paleness, jaundice, dark-colored urine, possible heart	Early symptoms are specific and include fatigue, arthralgia, impotence, abdominal pain, and weight	They are often the first set of manifestations and include headache, vertigo, dizziness, tinnitus, and

Clinical Manifestations	murmur, difficult to do physical activity, fever, and weakness.	loss. Later in the disease, the excess iron accumulates in the liver and causes liver enlargement and eventually cirrhosis. Excess iron can also lead to diabetes, skin bronzing, heart problems (cardiomyopathy), arthritis, and testicular atrophy. Symptoms usually don't develop until age 40 in men and after 50 in women.	visual changes. Circulatory manifestations occur due to the HTN caused by hypervolemia and hyperviscosity. The patient may have angina, HF, intermittent claudication, and thrombophlebitis and these manifestations are caused by impaired blood flow, vessel distention, circulatory stasis, thrombosis, and tissue hypoxia from the hypervolemia and hyperviscosity. Generalized pruritus (often worsened by a hot bath) may be a distinguishing symptom and is related to histamine release from an increased number of basophils. Paresthesia and erythromelalgia (painful burning and redness of the hands and feet) may be present.
Diagnostic Studies	Increased Hgb/Hct, normal to increased MCV, increased reticulocytes, normal to increased serum iron, normal to decreased TIBC, normal to increased ferritin, and increased bilirubin.	Laboratory values show a high serum level, 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.	Bone marrow examination in polycythemia vera shows hypercellularity of RBCs, WBCs, and platelets. Decreased to normal EPO level (secondary polycythemia has a high level). Increased RBC and Hgb count with microcytosis. Increased platelet count dysfunction

			Increased leukocyte alkaline phosphatase, uric acid, and cobalamin levels. High histamine levels. Increased WBC count with basophilia and neutrophilia.
Drug Therapy -include name, dosage, route, and nursing interventions	Folate replacement to suppress the RBC destruction. While taking folic acid supplements, monitor plasma folic acid levels, hemoglobin, hematocrit, and reticulocyte count before and periodically during therapy. Also, assess patients for signs of megaloblastic anemia (fatigue, weakness, dyspnea) before and periodically throughout therapy. Immunosuppressive agents may be used (glucocorticoids or rituximab) which is a monoclonal antibody to B-Cell CD20. For severe cases associated with hemolysis, thrombocytopenia, and acute kidney injury, plasma exchange and eculizumab, a monoclonal antibody to complement protein C5, can be used.	The goal of treatment is to remove excess iron from the body and minimize any symptoms. The blood is removed less often to maintain iron levels within normal limits Iron removal is achieved by removing 500mL of blood each week for 2-3 years until the iron stores in the body are depleted. Iron chelating agents may be used. Deferoxamine, which chelates and removes accumulated iron via the kidneys, can be given IV or SQ. Monitor for iron poisoning, changes in VS, decreased vision and hearing, I/Os and urine color.	Phlebotomy is the most important treatment. Treatment is directed towards reducing blood volume and viscosity and bone marrow activity. The aim of phlebotomy is to reduce the hematocrit and keep it less then 45%. At the time of diagnosis, 300-500mL of blood may be removed every few days. After that, phlebotomy may be needed every 2-3 months. Avoid iron supplementation and include adequate hydration. Low dose aspirin is given to prevent clots. Hydroxyurea, busulfan, and chlorambucil may be given. Anagrelide, which can reduce the platelet count and inhibit platelet aggregation, may be given. Allopurinol 150 or 300 mg PO may be given to reduce the number of acute gout episodes. While taking allopurinol, monitor I/Os to avoid toxicity, maintain

			adequate fluid intake, and monitor for joint pain and swelling.
Nursing Management -include patient education	Hemolytic crisis is a potential consequence so be ready to institute appropriate emergency therapies such as aggressive hydration and electrolyte replacement to reduce the risk for kidney tubules and shock. Supportive care includes giving corticosteroids and blood products or removing the spleen. General supportive care until the causative agent can be eliminated or at least made less injurious to the RBCs.	Teach patients that iron accumulation can be reduced by dietary changes, such as avoiding vitamin C and iron supplements, uncooked seafood, and other iron rich food. Management of organ involvement (diabetes, HF). With early diagnosis and treatment, life expectancy is normal but many cases go undetected and untreated.	Assist with performing phlebotomy Teach patients to start active and passive leg exercises and ambulation when possible to decrease thrombus formation. Teach patients to assess I/O during hydration therapy to avoid fluid overload or deficit. Assess patients nutritional status and for any complications.

List 3 effects that aging has on the Hematologic System

1. Decline in bone marrow cellularity.
2. Decreased lymphocyte count in both men and women.
3. Decreased basophil count in women.