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.	Group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production. Due to an absent or reduced globulin protein. a-Globin chains are absent or reduced in a-thalassemia, and B-globin chains are absent or reduced in B- thalassemia Genetic Link	Most common cause: pernicious anemia. It is caused by an absence of intrinsic factor (IF) -Insidious onset -begins in middle age or later (usually after age 40) -without IF, cobalamin will not be absorbed. - In pernicious anemia: gastric mucosa is not secreting IF Decrease of HCL causing decreased secretion of IF -In autoimmune process: antibodies are directed against the gastric parietal cells and /or IF and they are destroyed	Causes of folic acid deficiency -chronic alcoholism -chronic hemodialysis -dietary deficiency -Drugs: interfering with absorption or use of folic acid -Methotrexate -Antiseizure drugs (phenobarbital, phenytoin) -Increased requirement (Pregnancy) -Malabsorption syndromes: Celiac, Crohn's, small bowel resection
Clinical Manifestations	(In early course of iron-deficiency anemia) – May not be symptomatic. Chronic – Palpitations, mild fatigue, Exertional dyspnea Pallor (most common) Glossitis (2nd most) Cheilitis Headache, paresthesia, and a burning sensation of the tongue	Patient with thalassemia minor is often asymptomatic. Mild to moderate anemia with microcytosis(small cells) and hypochromia(pale cells), mild splenomegaly, bronzed color of skin, and bone marrow hyperplasia Thalassemia major is a life-threatening	Manifestation of anemia caused by tissue hypoxia. GI: sore, red, beefy, and shiny tongue; anorexia, nausea, and vomiting; abdominal pain Neuromuscular: weakness, paresthesia of feet and hands, reduced vibratory and position senses, ataxia muscle weakness and impaired thought (Confusion)	Similar to those of cobalamin deficiency. Develops insidiously. Symptoms maybe contributed to coexisting problems. GI: stomatitis, cheilosis, dysphagia, flatulence, and

		disease in which growth, both physical and mental, is often slowed. The person with thalassemia major is pale and displays other general symptoms of anemia. Develops in childhood by 2 years of age and can cause growth and developmental deficits. Jaundice from the hemolysis of RBCs is prominent. There is pronounced splenomegaly since the spleen continuously tris to remove the damaged red cells. Hepatomegaly and cardiomyopathy may occur from iron deposition		diarrhea. Can cause neurological symptoms.
Diagnostic Studies	Hgb, Hct - LOW MCV – LOW Reticulocytes – N or slight high or low Serum iron – LOW TIBC – HIGH Transferrin – N or LOW Ferritin – LOW Bilirubin – N or low Stool occult blood test Endoscopy colonoscopy	Thalassemia major: Hgb, Hct – LOW MCV – N or low Reticulocytes – HIGH Serum iron – HIGH TIBC – LOW Transferrin – LOW Ferritin – N or high Bilirubin – HIGH Folate – LOW	Hgb, Hct – LOW Reticulocytes- N or low Serum iron N or high Transferrin- slightly high Ferritin- HIGH Bilirubin- N or slightly high Serum B 12 – LOW RBC- appear macrocytic. Serum test – anti-IF antibodies GI endoscopy/ biopsy Serum methylmalonic acid (MMA) – HIGH Serum homocysteine - HIGH	Hgb, Hct – LOW MCV – HIGH Rect – N or low Serum iron- N or high Transferrin – slightly high Ferritin – HIGH Bilirubin – N to slight high Folate - LOW
Drug Therapy	Oral iron (enteric-coated or sustained-release capsules) - Daily dose 150-200mg of	No specific drug or diet therapies are effective. Thalassemia minor:	Parenteral vitamin B12 (cyanocobalamin, hydroxocobalamin) or intranasal cyanocobalamin	Replacement therapy: 1mg/day PO Chronic

	elemental iron Undiluted liquid iron Parenteral iron (IM or IV) Sodium ferrous gluconate and iron sucrose (alternatives) Extreme cases: Transfusion of pack RBCs	No treatment necessary Thalassemia major: Blood transfusion or exchange transfusions in conjunction with chelating agents to bind to iron. Drugs used during therapy: oral deferasirox (Exjade, JadeNu) or deferiprone (Ferriprox, or IV or subcutaneous deferoxamine (Desferal)	(Nascobal) 1,000 mcg/day of cobalamin IM for 2 weeks and then weekly until the hemoglobin is normal and then monthly for life Pts. With GI absorption: High – dose oral cobalamin and sublingual cobalamin are options.	alcoholism / Malabsorption: 5mg/day PO
Nursing Management	Goals ❖ Treat underlying problem that is causing iron loss or reduced intake (malnutrition , alcoholism) ❖ Replacing iron - Teach patient which foods are good sources of iron (Lean, beef, turkey, pork, chicken, fish, legumes dark green leafy vegetables, whole-grain and enriched bread and cereals, beans) - Avoid taking iron supplements with food	Thalassemia major: Chelating therapy ❖ Give Zinc & ascorbic acid, folic acid, supplements during chelation therapy. ❖ Iron should not be given. ❖ Ascorbic acid should only be taken with chelation because it increases the absorption of dietary iron. ❖ Monitor hepatic, heart, and lung function and treat as needed	❖ Tx manifestations of anemia ❖ Assess neurological difficulties that are not fully corrected by replacement therapy. ❖ Implement measure 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. ❖ Neuromuscular complications may not be reversible and physicial therapy may be needed.	❖ Teach patients to eat foods high in folic acid (Green leafy veg, enriched grain products and breakfast cereals, orange juice, peanuts, avocado

	- Iron should be taking 1 hour before meals. - Taking iron with orange juice or ascorbic acid increases the absorption of iron - Undiluted liquid iron may stain the patient's teeth therefore should be diluted and ingested through a straw - GI side effects may occur, have the patient stay upright for 30minutes after taking oral forms - Teach patient Iron preparations will cause their stools to become black. - Constipation is common, patient should start stool softeners or laxatives if needed.	Hematopoietic stem cell transplantation (HSCT) -disadvantages outweighs benefits -May require splenectomy		

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
	Caused by cancer,	Disease in which the	Sudden hemorrhage	Sources of chronic

Etiology	autoimmune and infectious disorders (HIV, hepatitis, malaria), HF, or chronic inflammation. Associated with an underproduction of RBCs and mild shortening of RBC survival. RBCs normocytic, normochromic, and hypo proliferative Cytokines release in these conditions (IL-6) cause an increased uptake and retention of iron within macrophages. This leads diversion of iron from circulation into storage sites with limited iron for erythropoiesis. (Cytokine dysregulation)	patient has peripheral blood pancytopenia (decrease of all blood cell types -RBCs, white blood cells WBC, and platelets and hypocellular bone marrow Rare incidence 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. Maybe acquired from toxic injury to bone marrow stem cells or result from an inherited stem cell defect	Causes include trauma, complications of surgery and conditions or diseases that disrupt vascular integrity. 1st Sudden reduction in the total blood volume can lead to hypovolemic shock. 2nd if acute loss is more gradual: body maintains its blood volume by slowly increasing the plasma volume; although these preserves circulating fluid volume, the number of RBSs available to carry O2 is significantly decreased	blood loss are similar to those of deficiency anemia (bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss) 3rd major cause of anemia is hemolytic anemia – destruction or hemolysis of RBCs at a rate that exceeds production
Clinical Manifestations	Similar manifestations as anemia; must be distinguished from other causes of anemia.	Can manifest abruptly (over days) or insidiously. Patient may have symptoms caused by suppression of any or all bone marrow elements. General manifestations of anemia (fatigue and dyspnea) Patient with neutropenia (low neutrophil count) Is susceptible to infection & a risk for septic shock and death. Thrombocytopenia is manifested by a predisposition to bleeding (nosebleed)	10% 500ml – None or rare vasovagal syncope 20% 1000ml- No detectable signs or symptoms at rest. Tachycardia with exercise and slight postural hypotension 30% 1500ml- Normal supine blood pressure and pulse at rest. Postural hypotension and tachycardia with exercise 40% 2000ml- BP, central venous pressure, and cardiac	General manifestations of iron deficiency anemia

			output below normal at rest; air hunger; rapid, thready pulse and cold, clammy skin 50% 2500ml- Shock, lactic acidosis, and potential death	
Diagnostic Studies	Serum ferritin – HIGH Increased iron stores distinguish it from iron-deficiency anemia. Normal folate and cobalamin blood levels distinguish it from megaloblastic anemias from folate and cobalamin deficiencies.	Lab studies confirm diagnosis. Hgb, WBC, &platelet – Low Reticulocyte – LOW Serum iron and TIBC – may be high. Bone marrow biopsy, aspiration, and pathologic examination maybe done	RBC value may be N or high for 2 to 3 days. Once plasma volume is replaced, the RBC mass is less concentrated then RBC, hemoglobin, & hematocrit levels - LOW	Hgb, Hct – LOW MCV – LOW Reticulocytes – N or high Serum iron – LOW TIBC – LOW Bilirubin- N or low
Drug Therapy	Correct underlying disorder. If severe: blood transfusion maybe needed. Erythropoietin therapy is used for anemia related to renal disease and cancer (Its use is limited because of increased risk for thromboembolism and death in some patients)	HSCT (Tx of choice for younger patients 55 & under who do not responds to immunosuppressive therapy) and immunosuppressive therapy with antithymocyte globulin , steroids, and cyclosporine or cyclophosphamide. Eltrombopag (Promacta) (For older adults without a donor) High dose of cyclophosphamide, alemtuzumab, or androgens maybe helpful in select patients who do not respond to other therapies.	IV fluids used in emergencies include dextran, hetastarch, albumin, and crystalloid electrolyte solutions (lactated Ringer's) Blood transfusion (packed RBCs) Large amt. of blood: Platelets, Plasma, and cryoprecipitate may be needed.	same as iron deficiency anemia
Nursing Management	❖ Treat underlying disorder	❖ Management of aplastic anemia is based on identifying and removing the causative agent	❖ Interprofessional car is initially concerned with 1. Replacing	❖ Management of chronic blood loss anemia involves

		and providing supportive care until pancytopenia reverses. ❖ Nursing interventions to pancytopenia, thrombocytopenia, and neutropenia ❖ Prevent complications from infection and hemorrhage.	blood volume to prevent shock. 2. Finding the source of the hemorrhage and stopping the blood loss ❖ Assess the patient's expression of pain (pain in abdomen maybe numb and pain in lower extremity from compression of the lateral cutaneous nerve) ❖ Patient may need supplemental iron ❖ Post op patients: carefully monitor blood loss	identifying the source and stopping the bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Caused by destruction or hemolysis of RBCs at a rate that exceeds production. (antibodies or Infectious agents) Hemolytic anemia can occur because of problems intrinsic and extrinsic to the RBCs. Intrinsic hemolytic anemias, which are usually hereditary, result from defects in the RBCs themselves. In this type of anemia, the RBCs are normal, but	-iron overload disorder -Genetic, auto recessive disorder - accumulation of iron at an increased rate and may exceed total iron concentrations of 50g	-production & presence of increased numbers of RBCs. 2 types: primary (polycythemia vera) or secondary. Primary: chronic myeloproliferative disorder, involves RBCs, WBCs, & platelets. Increases leads to enhanced blood viscosity & blood volume which causes congestion of organs and tissues.

	external factors are causing damage.		-Genetic link Secondary: can be either hypoxia driven or hypoxia independent. Hypoxia driven: hypoxia stimulates erythropoietin production in the kidneys, which in turn stimulates RBC production. Levels may return to normal after body is done compensating and normal O2 levels resume. Hypoxia independent: cancer or benign tumor tissues makes EPO. Serum EPO stays increased. Splenomegaly does not occur.
Clinical Manifestations	General manifestations of anemia Jaundice Enlargement of Spleen & liver	Do not develop until after age 40 in men & 50 in women. Early: fatigue, arthralgia, impotence, abdominal pain, and weight loss Late: Liver enlargement; eventually cirrhosis Diabetes, skin pigment changes (bronzing), heart problems, arthritis and testicular atrophy.	Polycythemia vera: 1st manifestations: headache, vertigo, dizziness, tinnitus, and visual changes Generalized pruritus. Paresthesia Erythromelalgia May have angina, HF, intermittent, claudication, and thrombophlebitis which may be embolization. Hemorrhagic phenomena Satiety and fullness (hepatomegaly & splenomegaly) Pain Plethora (ruddy complexion) Hyperuricemia & Gout
Diagnostic Studies	Hgb, HCT – LOW MCV- N or high Reticulocytes – HIGH Serum iron – N or high TIBC – N or low Ferritin – N or high Bilirubin - HIGH	High serum iron, TIBC, and serum ferritin. Test for genetic mutations confirms diagnosis. Liver biopsy	Polycythemia vera: 1) high hemoglobin and RBC count with microcytosis 2) low to normal EPO level (secondary polycythemia has a high level) 3) high WBC count with basophilia and neutrophilia 4) high platelet count

			(thrombocytosis) and platelet dysfunction 5) high leukocyte alkaline phosphatase, uric acid, and cobalamin levels 6) high histamine levels Bone marrow examination in polycythemia vera shows hypercellularity or RBCs, WBCs, & platelets
Drug Therapy	Emergency therapy: aggressive hydration and electrolyte replacement to reduce the risk for kidney injury caused by hemoglobin (from RBC lysis) Severe Cases: Folate therapy Immunosuppressive agents: glucocorticoids or rituximab (Rituxan) Extreme Cases: Plasma exchange and eculizumab (Soliris)	Goal of treatment: Remove excess iron. -removed by removing 500ml of blood each week for 2 to 3 years until the iron stores in the body are depleted Iron chelating agents may be used (Deferoxamine IV or subcutaneously)	Phlebotomy to reduce hematocrit. Hydration therapy Myelosuppressive agents (hydroxyurea, busulfan (Myleran), and chlorambucil (leukeran) Ruxolitinib (Jakafi) for patients who have not responded to hydroxyurea. Anagrelide (Argrylin) Low dose aspirin a-interferon & pegylated interferon Allopurinol (Zyloprim)
Nursing Management	❖ Supportive care until the causative agent can be eliminated. ❖ Preserve renal function.	❖ Make appropriate dietary changes, reduction in iron (avoid vitamin C, iron supplements, uncooked seafood, and iron rich foods) ❖ Management of organ involvement (diabetes, HF)	❖ Teach patient avoid iron supplementation during phlebotomy treatments. ❖ Assist with or perform the phlebotomy. ❖ Assess fluid intake and output during hydration therapy to avoid fluid overload/deficit. ❖ Give myelosuppressive agents as ordered. ❖ Observe the patient and teach the patient about the side effects of the drugs. ❖ Assess the patient's nutritional status.

			❖ Begin activities and/or medications to decrease thrombus formation. ❖ Start active or passive leg exercises and ambulation when possible. ❖ Assess patient for complications