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.	A group of diseases involving inadequate production of normal hemoglobin, which decreases RBC production. Due to an absent or reduced globulin protein. Commonly found in members of ethnic groups whose origins are near the Mediterranean Sea and equatorial or near-equatorial regions of Southern Asia, the Middle East, India, Pakistan, China, Southern Russia, and Africa.	The most common cause of cobalamin deficiency is pernicious anemia. It is caused by an absence of intrinsic factor. It begins in middle age or later (usually after age 40) with 60 years being the most common age at diagnosis. Without IF cobalamin will not be absorbed. There is also a familial predisposition for pernicious anemia, so evaluate patients who have a positive family history of pernicious anemia.	causes megaloblastic anemia. Needed for DNA synthesis leading to RBC formation and maturation, Common causes: chronic alcoholism, malabsorption syndromes, dietary deficiency, chronic hemodialysis
Clinical Manifestations	Early on in iron-deficiency anemia, the patient may not have symptoms. As the disease becomes chronic, general manifestations include pallor, glossitis, and cheilitis. The patient may report headaches, paresthesia, and a burning sensation of the tongue, all of which are caused by a lack of iron in the tissues.	Thalassemia Minor • often asymptomatic • mild to moderate anemia with microcytosis (small cells) and hypochromia (pale cells) • mild splenomegaly • bronzed color of skin • bone marrow hyperplasia Thalassemia Major • physical and mental growth slowed • pale • growth/ developmental deficits • other general anemia symptoms Jaundice, Pronounced splenomegaly, Hepatomegaly, Cardiomyopathy	General manifestations of anemia related to cobalamin deficiency develop because of hypoxia. GI manifestations include 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 process ranging from confusion to dementia.	Symptoms may be attributed to other coexisting problems such as cirrhosis/esophageal varices GI Issues: • stomatitis • cheilosis • dysphagia • flatulence • diarrhea • neurologic symptoms
Diagnostic Studies	• Hgb/Hct= decreased • MCV=decreased • -Reticulocytes= N or slightly increased or decreased • -Serum Iron (decreased) • -TIBC= increased • -Transferrin= N or decreased • -Ferritin= decreased • -Bilirubin= N or decreased • -Serum B12= N • -Folate= N • Stool Occult Blood Test • Endoscopy/Colonoscopy • Bone Marrow Biopsy	• Hgb/Hct- decreased • MCV- N or decreased • Reticulocytes- increased • Serum Iron- increased • TIBC- decreased • Transferrin- decreased • Ferritin- N or increased • Bilirubin- increased • Serum B12- N	• Hgb/Hct- decreased • MCV- increased • Reticulocytes- N or decreased • Serum Iron- N or increased • TIBC- N • Transferrin- slight increase • Ferritin- increased • Bilirubin- N or	• Hgb/Hct- decreased • MCV- increased • Reticulocytes -normal of decreased • Serum Iron- normal or increased • TIBC- normal • Transferrin-

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 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	It is associated an underproduction of RBCs and mild shortening of RBC survival	Caused by autoimmune activity by autoreactive T lymphocytes. The cytotoxic T cells target and destroy the patient's own hematopoietic stem cells.	Acute blood loss occurs because of sudden hemorrhage Causes of acute blood loss include trauma, complications of surgery, conditions/diseases that disrupt vascular integrity, hypovolemic shock & a decrease in RBC's caring O2 are clinical concerns	The effects of chronic blood loss are usually related to the depletion of iron stores and considered an iron deficiency anemia.
Clinical Manifestations	Usually develops after 1 to 2 months of disease activity and has an immune basis. Interkeukin-6 causes an increased uptake and retention of iron within macrophages.	Aplastic anemia can manifest abruptly over days or insidiously over weeks to months. Can vary from mild to severe. General manifestations: • Fatigue, dyspnea, cardiovascular, cerebral responses • low neutrophil count is susceptible to infection and at risk for shock and death.	• 10%(500mL)= none or rare vasovagal syncope • 20%(1,000mL)= No detachable s/s at rest. Tachycardia w/ exercise and slight postural hypotension • 30%(1,500mL)= normal supine blood pressure and pulse at rest. Postural hypotension and tachycardia w/ exercise • 40%(2,000mL)= BP, central venous pressure, and cardiac output below normal at rest; air hunger; rapid/thready pulse, and clod/clammy skin • 50%(2,500mL)= shock, lactic acidosis, &	The sources of chronic blood loss are similar to those of iron deficiency anemia like bleeding, ulcer, hemorrhoids, menstrual and postmenopausal blood loss.

			potential death	
Diagnostic Studies	Normal folate and cobalamin blood levels distinguish it from megaloblastic anemias from folate and cobalamin deficiencies.	• Hgb/Hct- decreased • MCV- N or increased • Reticulocytes- decreased • Serum Iron- N or increased • TIBC-N or increased • Transferrin- N • Ferritin- N • Bilirubin- N • Serum B12- N • Folate- N	• Hgb/Hct- decrease • MCV- N or decreased • Reticulocytes- N or increased • Serum Iron- N • TIBC- N • Transferrin- N • Ferritin- N • Bilirubin-N • Serum B12- N • Folate-N	• Hgb/Hct- decreased • MCV- decreased • Reticulocytes- N or increased • Serum Iron- decreased • TIBC- decreased • Transferrin- N • Ferritin- N • Bilirubin- N or decreased • Serum B12- N • Folate- N
Drug Therapy	Correct the underlying disorder. If anemia is severe blood transfusions may be needed, but they are not recommended for long term treatment.	Management is based on identifying and removing the causative agent when possible and providing supportive care until the pancytopenia reverses	IV fluids for emergencies including dextran, etastarch, albumin, crystalloid electrolyte solutions (ex. lactated ringers' solution), Blood transfusions (Packed RBCs), May give platelets /plasma/cryoprecipitate, supplemental iron (oral or parenteral)	Supplemental iron may be needed.
Nursing Management	No nursing interventions mentioned in the section	Directed at preventing complications from infection and hemorrhage	For postoperative pt carefully monitor the blood loss from various drainage tubes/dressings and implement appropriate actions, giving blood products.	Management of chronic blood loss anemia involves identifying the source of stopping bleeding.

Table 3	**Acquired Hemolytic Anemia**	**Hemochromatosis**	**Polycythemia**
Etiology	Results from hemolysis of RBCs from extrinsic factors. Factors include physical destruction, antibody reactions, and	Iron overload disorder. Genetic defect is most common cause. It is also caused by sideroblastic anemia/liver	Production and presence of increased numbers of RBCs. The increase in RBCs can be so great that blood circulation is impaired because of the

	infectious agents and toxins.	disease/and chronic blood transfusions.	increased blood viscosity and volume. Two types of primary polycythemia and secondary polycythemia.
Clinical Manifestations	Dyspnea, angina. Tachycardia, malaise, dizziness.	• s/s develop after age 40 in men and after age 50 in women • early s/s are nonspecific & include: fatigue, arthralgia, impotence, abdominal pain, weight loss, Later excess iron accumulates in liver and causes liver enlargement and cirrhosis, diabetes, skin pigment changes (bronzing), heart problems, arthritis, testicular atrophy	Headache, vertigo, dizziness, tinnitus, and visual changes. Generalized pruritus after a hot bath may be a striking symptom. may have burning and redness of hands and feet.
Diagnostic Studies	• Hgb/Hct- decreased • MCV- N or increased • Reticulocytes- increased • Serum Iron- N or increased • TIBC-N or decreased • Transferrin- N • Ferritin- N or increased • Bilirubin- increased • Serum B12- N • Folate- N	high serum iron, high TIBC, high serum ferritin, Testing for now genetic mutations confirms the diagnosis, Liver biopsy can establish the degree of organ damage	High hemoglobin and RBC count with microcytosis, low to normal EPO level (Secondary high), 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. Bone marrow examination in polycythemia vera shows hypercellularity of RBCs, WBCs, and platelets.
Drug Therapy	The patient with chronic hemolytic anemia may need folate replacement. To suppress RBC destruction, immunosuppressive agents may be used, such as glucocorticoids or rituximab.	• removing 500mL of blood each week for 2-3 years until the iron stores in the body are depleted • iron chelating agents (Deferoxamine, IV or	Low dose aspirin is used to prevent clotting. Avoid iron supplements. Hydration therapy can reduce the bloods viscosity. Anagrelide can reduce the platelet count and inhibit platelet aggregation. Pegylated interferon are given

		subcutaneously) • Deferasirox and Deferiprone(oral agents)	to women of childbearing age or those with intractable pruritus. Allopurinol may reduce the number of acute gouty attacks.
Nursing Management	Treatment and management of acquired hemolytic anemias involve general supportive care until the causative agent can be eliminated or at least made less injurious to the RBC's.	Teach pt to avoid vitamin C, iron supplements, uncooked seafood/ and iron rich foods.	Treatment is directed toward reducing blood volume and viscosity and bone marrow activity. Phlebotomy is the mainstay of treatment.