

Alicia McGraw
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 B₁₂) Deficiency	Folic Acid Deficiency
Etiology	Inadequate dietary intake, malabsorption, blood loss, or hemolysis.	Inadequate production of normal hemoglobin, autosomal recessive genetic basis, Commonly found in ethnic groups near Mediterranean sea, SE asia, middle east, india, Pakistan, china, southern Russia or Africa.	Pernicious anemia – absence of intrinsic factor. Chronic alcoholism, dietary deficiency, pregnancy, intestinal malabsorption.	Chronic alcoholism, chronic hemodialysis, dietary deficiency, drugs interfering with absorption or use of folic acid, celiac disease, crohns disease, small bowel resection.
Clinical Manifestations	Lethargy, pale skin, tachypnea, systolic murmur, abdominal distention, headache, confusion, irritability.	Often asymptomatic, pale, growth and developmental deficits, jaundice, splenomegaly, hepatomegaly and cardiomyopathy,	Tissue hypoxia- sore red and beefy tongue, anorexia, nausea and vomiting. Weakness paresthesia’s of feet, impaired thought process.	Similar to those of Cobalamin deficiency.
Diagnostic Studies	Hgb/Hct, MCV, Transferrin, Bilirubin.	CBC.	Serum folate levels, anti-IF antibodies, biopsy of GI mucosa.	Serum folate levels, Hgb/Hct, MCV, Serum iron, etc.
Drug Therapy	Oral iron, IV iron, vitamin C.	Minor does not need treatment. Major is treated with blood transfusions, in conjunction with chelating agents that bind to iron.	Parenteral vitamin B12 or intranasal cyanocobalamin.	Replacement therapy with folic acid.
Nursing Management	Treat the underlying problem causing the anemia, replace the iron.	Educate on treatment of disease.	Assess for neuro deficits that aren’t corrected by drug therapy, protect from falls burns and trauma.	Teach patients to eat food high in folic acid-green leafy veggies, enriched grains and breakfast cereal, orange juice peanuts, avocados.

Table 2	Anemia of Chronic Disease	Aplastic Anemia	Acute Anemia due to blood loss	Chronic Anemia due to blood loss
Etiology	Cancers, autoimmune disease, infectious disorders (HIV, hep, malaria)	Autoimmune activity by autoreactive T lymphocytes.	Sudden hemorrhage.	Bleeding ulcer, hemorrhoids, menstrual and postmenopausal blood loss.
Clinical Manifestations	Underproduction of RBCs and shortening of RBC survival. Manifestations of whatever the causing factor is.	Abruptly or over months. Fatigue, dyspnea, infections.	Syncope, postural hypotension, tachycardia, low CO, air hunger, rapid thready pulse, shock, lactic acidosis, potential death.	Weakness, fatigue
Diagnostic Studies	Serum ferritin levels and increased iron stress, folate levels and cobalamin.	Hgb/Hct, WBC, platelets, RBC, bone marrow biopsy.	Lab results will not be accurate right away, after a few days can be seen in RBC, Hemaglobin and hematocrit levels low.	CBC, iron levels
Drug Therapy	Correct underlying disorder. Blood transfusions.	Identify and remove causative agent, supportive care until pancytopenia reverses. Immunosuppressive therapy with antithymocyte globulin, steroids, cyclosporine.	IV fluids such as dextran, hetastarch, albumin and crystalloid electrolyte solutions. Packed RBCs. Supplemental iron.	Packed RBCs
Nursing Management	Educate on diet and medication treatments.	Prevent complications from infection and hemorrhage.	Replace blood volume, prevent shock, find source of hemorrhage.	Identify and stop source.

Table 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	External factors cause damage to RBCs. Macrophages in spleen, liver, and bone marrow destroy RBCs that are old, defective, or moderately damaged.	Caused by a genetic defect, may also occur with other diseases such as sideroblastic anemia. Liver disease and chronic blood transfusions used to treat thalassemia and SCD.	Genetic mutation with primary, hypoxia in secondary.

Clinical Manifestations	Same manifestations as anemia. Jaundice is likely. Splenomegaly and hepatomegaly likely.	Develop later in life, fatigue, impotence, abdominal pain and weight loss, cirrhosis, heart problems, DM.	HTN caused by hypervolemia and hyperviscosity. Dizzy, headache, vertigo, tinnitus and visual changes.
Diagnostic Studies	Lab levels such as CBC, Hgb/Hct, anemia levels, kidney function labs.	Serum iron, TICB, serum ferritin, genetic testing, liver biopsy.	Hemoglobin, CBC, EPO levels, WBC count, platelet count, uric acid, leukocyte alkaline phosphatase, cobalamin levels.
Drug Therapy	Glucocorticoids such as prednisone.	Iron chelating agents, dietary changes such as avoiding vitamin c and iron supplements, uncooked seafood and iron rich foods.	Phlebotomy is mainstay of treatment. Hydration therapy, myelosuppressive agents, anagrelide, low dose aspirin.
Nursing Management	Maintain renal function.	Management of organ involvement.	Assist with phlebotomy, assess fluid intake, teach patient about side effects of drug therapy, assess nutritional status, leg exercises when possible.