Unit 7: Hematology
Chapter 29 & 30
ONLINE CONTENT (1.5 H)
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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	Develops because of inadequate dietary intake, malabsorption, blood loss, or hemolysis.	Group of diseases involving inadequate production of normal hemoglobin which decreases RBC production. It is due to reduced globulin protein.	The gastric mucosa is not secreting IF because of either gastric mucosal atrophy or autoimmune destruction of parietal cells	Folic acid deficiency also causes megaloblastic anemia. Folic acid is needed for DNA synthesis leading to RBC formation and maturation.
Clinical Manifestations	May not have symptoms as the disease becomes chronic manifestations of chronic anemia can be present most common glossitis and second most common cheilitis.	Patients with thalassemia minor is asymptomatic, patient that has mild or moderate anemia with microcytosis (small cells) and hypochromia (pale cells), mild splenomegaly, bronzed color of the skin and bone marrow hyperplasia. This is life threatening disease in which growth, both physical and mental is both slowed down.	General manifestations of anemia. GI- Sore, red, beefy, and shiny tongue, anorexia, N/V, abdominal pain, neuromuscular-weakness, paresthesia of the feet and hands, reduced vibratory and position senses, ataxia, muscle weakness, and impaired thought process ranging from confusion to dementia.	General manifestations of anemia. GI- stomatitis, cheilosis, dysphasia, flatulence and diarrhea. Thiamine deficiency can cause neurological problems
Diagnostic Studies	Stool for occult blood, endoscopy, colonoscopy, bone marrow biopsy, Hgb/Hct, MCV, reticulocytes, Serum Iron, TIBC, Ferritin, bilirubin, Serum B_{12} Folate, blood test, history and physical.	Hgb/Hct, MCV, Reticulocytes, Serum Iron, TIBC, Ferritin, Bilirubin, Serum $B_{12,}$ Folate	Hgb/Hct, MCV, Reticulocytes, Serum B_{12}, Folate, RBC appear larger and have abnormal shapes, Serum ant-IF test, Upper GI endoscopy, Serum methylmalonic acid, Serum homocysteine	Hgb/Hct, MCV, Reticulocytes, Serum Iron, TIBC, Ferritin, Bilirubin, Serum $B_{12,}$ Folate, Serum Folte level is low with normal serum cobalamin level

Drug Therapy	Oral iron, Ferrous sulfate or ferrous gluconate. I.V: iron dextran, sodium ferrous gluconate, iron sucrose, Transfusion of RBC's.	Minor- No specific drug therapy. Major- Blood transfusion or exchange transfusions in conjunction with chelating agents. P.O- deferasirox or deferiprone. IV- or Subcu- deferoxamine. Ascorbic acid taken with chelation, Folic acid, zinc supplements.	Parental vitamin $B_{12,}$ or intranasal cyanocobalamin 1000 mcg/day of cobalamin IM for 2 weeks then monthly for life.	Folic Acid replacement therapy 1mg/day PO up to 5mg/day if suffer from chronic alcoholism
Nursing Management	Main goal is to treat underlying problem that is causing iron loss and reduce intake (eg. Malnutrition, alcoholism,) or poor absorption of iron. Patient may need blood transfusion of packed RBC's.	Monitor hepatic, heart and lung function	Assess neurologic difficulties, reduce risk of injury due to reduced sensitivity to heat and pain. Protect pt from falling, burns and trauma.	Teach patients to eat food high in folic acid (green leafy vegetables, enriched grains, breakfast cereals, orange juice, peanuts and avocado.

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to blood loss**	**Chronic Anemia due to blood loss**
Etiology	Associated with underproduction of RBC's and mild shortening of RBC survival. Develops 1-2 months after disease activity, has an immune basis.	Due to autoimmune activity by autoreactive T lymphocytes, The cytotoxic T cells target and destroy the patients own hematopoietic stem cells.	A sudden hemorrhage caused by trauma, complications of surgery, and conditions or diseases that disrupt vascular integrity	Depletion of iron stores and considered an iron deficiency anemia
Clinical		Manifest abruptly or insidiously. General manifestations such	500ml-none or vasovagal syncope 1000ml-no S/S at	Bleeding ulcer, hemorrhoids, menstrual and

Manifestations	Chronic disease (HIV, Hepatitis, malaria), HF, or chronic inflammation, mild anemia (palpitations, fatigue, exertional dyspnea)	as fatigue, dyspnea, Thrombocytopenia, neutropenia.	rest, tachycardia with exercise and slight postural hypotension 1500ml-postural hypotension and tachycardia with exercise. 2000ml-BP, central venous pressure and cardiac output below normal at rest; air hunger; rapid thready pulse and cold clammy skin 2500ml-shock, lactic acidosis and potential death.	postmenopausal blood loss.
Diagnostic Studies	High serum ferritin and increased iron stores. Normal folate and cobalamin blood levels	Hgb, WBC, and platelet values are decreased. RBC indices are normal. Reticulocyte count is low. Serum iron and total iron-binding may be high. Bone marrow biopsy, aspiration pathologic examination.	RBC's Hgb/ Hct levels are low	
Drug Therapy	No long-term treatment. If severe may need blood transfusion. Erythropoietin therapy is used for anemia related to renal disease.	HSCT and immunosuppressive therapy with antihymocyte globulin, steroids, cyclosporine, or cyclophosphamide	IV fluids; dextran hetastarch, albumin, and LR, Packed RBC's, platelets, plasma, and cryoprecipitate. Supplement iron	Supplemental iron
Nursing Management	Correct the underlying disorder. Blood transfusion if anemia is severe. Erythropoietin therapy r/t renal disease and cancer	Identify and remove causative agent. Prevent infection and hemorrhage	Monitor blood loss from various drainage tubes and dressings, implement appropriate actions. Giving blood products.	Identify the source and stopping the bleeding. May have to give supplemental iron

Table 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	Condition caused by the destruction or hemolysis of RBCs at a rate that exceeds production.	Iron overload disorder. May occur with disease such as sideroblastic anemia. It also may be caused by a liver disease and the chronic blood transfusion used to treat thalassemia.	The production and presence of increased numbers of RBC's this impairs circulation because of the viscosity and volume of the blood.
Clinical Manifestations	General manifestations of anemia. RBC's are normal but external factors are causing damage. Antibodies against RBC's, Jaundice Elevation in bilirubin levels, Enlarged spleen and liver. Antibody reactions, Infectious agents and toxins	Symptoms do not develop to after age of 40 in men and 50 in women, fatigue, arthralgia, impotence, abdominal pain, and weight loss. Liver enlargement, and eventually cirrhosis, cardiomyopathy, skin bronzing, arthritis, testicle atrophy.	Circulatory manifestations of polycythemia vera occur because of the hypertension, hypovolemia, hyperviscosity. This includes fatigue, HA, vertigo, dizziness, tinnitus, and visual changes.
Diagnostic Studies	Bilirubin levels, Hgb/Hct, MCV, Reticulocytes, serum iron, TIBC, Ferritin,	Serum iron, TIBC, serum ferritin,	Hgb, RBC with microcytosis, EPO levels, WBC with basophilia, neutrophilia, platelet, Uric acid, cobalamin levels,
Drug Therapy	Main focus is to maintain renal function. Folate replacement, corticosteroids, immunosuppressive agents such as glucocorticoids or rituximab	Iron chelating may be used, Deferoxamine which chelates and removes accumulated iron via kidneys, can be given IN or subQ.	Myelosuppressive agents such as hydroxyurea, busulfan (myleran) and chlorambucil (Leukeran), low dose aspirin, allopurinol
Nursing Management	Hydration (aggressive), electrolytes replacement to reduce the risk of kidney injury,	Diet changes such as reduce vitamin c intake and avoid iron enriched foods.	Assess fluid intake and output during hydration therapy to prevent fluid overload, monitor for fluid deficit. Give myelosuppressive agents as

	Supportive care like giving corticosteroids, and blood products.		ordered and observe patient for side effects. Assess patients' nutritional status. Start active or passive leg exercise. Ambulation when possible. Requires ongoing evaluation. Assess the patient for complications.