Ashley Huntley

Unit 7: Hematology
Chapter 33 & 34
ONLINE CONTENT (2H)

Complete the worksheet and submit in the Unit 7: Hematology dropbox by March 18, 2024 at 0800. Please be sure to bring a copy to class on March 18, 2024.

Table 1	Iron Deficiency Anemia	Thalassemia	Cobalamin (Vitamin B_{12}) Deficiency	Folic Acid Deficiency
Etiology	May develop from inadequate intake, malabsorption, blood loss or hemolysis. Iron absorption occurs in the duodenum- GI surgeries may affect this. GI blood loss can be from peptic ulcers, gastritis, esophagitis, diverticula, hemorrhoids, and cancer. Postmenopausal women and dialysis pts. may also be at risk.	A group of diseases involving inadequate hgb production, which decreases RBC Production. Due to absent of reduced globulin protein. Hemolysis occurs. Common in ethnicities near the Mediterranean Sea and equatorial regions of southeastern Asia, middle east, India, Pakistan, China, Southern Russia, and Africa.	Gastric Mucosa does not secrete intrinsic factor, IF is required for cobalamin absorption in the distal ileum. GI surgeries, small bowel resections, Crohns, ileitis, celiac, diverticula, or chronic gastritis are at risk. Alcohol, hot tea, smoking, PPI and vegetarians are at increased risk. Patients with a family history are at risk.	Folic acid is needed for DNA synthesis needed for RBC formation and maturation. Chronic alcohol use, hemodialysis, diet deficiency, metformin, phenytoin use, pregnancy, Malabsorption syndromes.
Clinical Manifestations	Pallor is the most common finding, Glossitis (inflammation of the tongue) is the second most common. Cheilitis- inflammation of the lips, Headache, paresthesia, and burning sensation of the tongue.	T Minor- Asymptomatic- mild-moderate anemia, microcytosis (small cells) hypochromia (pale cells), mild splenomegaly, bronzed skin, bone marrow hyperplasia T Major-life-threatening. Develops in childhood by 2 pale and general symptoms of anemia, Jaundice, Pronounced splenomegaly, hepatomegaly and cardiomyopathy.	Tissue hypoxia leads to common anemia symptoms. Sore, red, beefy, shiny tongue. Anorexia, nausea, vomiting. Abdominal pain. Weakness, paresthesia of the feet and hands. Impaired cognition. Make take several months or years for symptoms to develop.	Similar to cobalamin deficiency. Develops insidiously. Stomatitis, cheilosis, dysphagia, flatulence, diarrhea. Thiamine deficiency. Neurological Symptoms.
Diagnostic Studies	Stool occult blood, endoscopy or colonoscopy, bone marrow biopsy as last resort. Hgb/Hct, MVC, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, B12, Folate.	Hgb/Hct, MVC, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, B12, Folate.	Hgb/Hct, MVC, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, B12, Folate. RBC are large and have abnormal shapes	Hgb/Hct, MVC, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, B12, Folate low. Normal serum cobalamin.
Drug Therapy	Oral Iron Supplements, Take with Vitamin C or orange juice. Take 1 hour before meals for best absorption.	Minor- no treatment needed. Major- Blood or exchange transfusion with chelating agents. Deferasirox, deferiprone, deferoxamine. Splenectomy. Hematopoietic Stem cell Transplant is the only cure. This	Parenteral vitamin b12 or intranasal cyanocobalamin. Typically, 1000 mcg/day or cobalamin IM for 2 weeks then weekly till hgb is normal. Then monthly for life.	Folic Acid replacement therapy 1-5 mg/day by mouth. Duration depends on severity.

		treatment comes with major risks and chelation therapy is proving to have better outcomes.		
Nursing Management	Identify and treat underlying cause, Drug therapy, Nutritional modifications, Packed RBC Transfusion.	Monitor liver, heart, and lung function. Administer medications. Give blood transfusions.	Nursing Care for anemia, assess for neurological problems, implement safety measures due to decreased sensitivity to heat and pain. Protect the patient from falls, burns, and trauma.	Teach patients to eat food high is folic acid (green leafy vegetables, enriched grain, breakfast cereals, orange juice, peanuts, avocado)

Table 2	**Anemia of Chronic Disease**	**Aplastic Anemia**	**Acute Anemia due to Blood Loss**	**Chronic Anemia due to Blood Loss**
Etiology	Also called anemia of inflammation. Develops after 1-2 months of disease activity. Cancer, autoimmune, infectious disorders, HF, or chronic inflammation. Bleeding disorders can contribute to anemia of chronic disease. Underproduction of RBC's and mild shortening of RBC survival. Iron is limited for RBC production	Decrease in all blood type cells-RBCs, WBCs, Platelets. And hypocellular bone marrow. Rather rare with 2 ppl/million in the US per year. 70% is due to autoimmune activity. T-Cells target hematopoietic stem cells. Also caused by toxin injury to bone marrow stem cells or an inherited stem cell defect.	Occurs with sudden bleeding, trauma, surgery. Body attempts to maintain blood volume by increasing plasma levels. This preserves circulating volume but the number of circulating RBCs available to carry oxygen decrease.	Blood loss similar to iron deficiency anemia- GI surgeries may affect this. GI blood loss can be from peptic ulcers, gastritis, esophagitis, diverticula, hemorrhoids, and cancer.
Clinical Manifestations	Similar to anemia	Can manifest abruptly or insidiously. Can be mild to severe. Fatigue and dyspnea, cardiovascular and cerebral responses. Neutropenia- susceptible to infection, risk for septic shock and death. Thrombocytopenia can lead to bleeding.	Caused by the bodies attempt to maintain adequate blood volume. At the most minimal(500mL)- none or minimal symptoms. At the most severe (2500mL)- Shock, lactic acidosis, and potential death. s/s are more important than lab values	Symptoms due to depleted iron stores. Considered an iron deficiency anemia.
Diagnostic Studies	High serum ferritin, increased iron stores distinguishes from iron deficiency anemia. Normal folate and cobalamin levels.	Hgb, WBC, plts. Are decreased. Reticulocyte low. Bone marrow biopsy, aspiration, & pathologic examination to confirm laboratory findings. Marrow has increased yellow marrow, high fat content.	Lab values will not reflect RBC loss. Values may seem normal or high for 2-3 days. Once plasma volume is replacing, RBC mass is less concentrated. Then RBC, Hgb, and Hct. Levels are low.	Imaging to identify the source of the blood loss.
Drug Therapy	Correct the underlying problem. If anemia is severe, blood transfusion. EPO for renal disease and cancer.	Immunosuppressant therapy, cyclosporine, Eltrombopag, cyclophosphamide, alemtuzumab, androgens, iron-binding agents.	Replace blood volume to prevent shock, promote coagulation to prevent further bleeding, find the source of bleeding and stop it. Blood product transfusions	Iron Supplements
Nursing Management	Administer medications related to underlying treatment and blood transfusions	Prevent complications from infection and bleeding. Blood transfusions.	Assess the pts pain, numbness. Monitor for shock	Identify the source of the bleeding and stop the bleeding.

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Table 3	Acquired Hemolytic Anemia	Hemochromatosis	Polycythemia
Etiology	Hemolysis of RBCs from extrinsic factors by physical destruction, antibody reactions, infectious agents and toxins. Traumatic events, Increased force of blood through abnormal veins and arteries, antibodies destroying RBCs. Autoimmune reactions also destroy RBCs.	Iron overload disorder with increased intestinal absorption. Genetic defect is the most common cause. Diseases such as sideroblastic anemia and liver disease, and chronic blood transfusions.	Production and presence of in numbers of red blood cells. Bl circulation can be impaired du increased viscosity and volum blood. Primary- Increased production but also WBCs and Platelets. I age of diagnosis is 60 and predominantly male. Secondary- hypoxia stimulates Increased EPO=more RBC pr May be from high altitude, lun cardiovascular disease, alveola hypoventilation, defective O2 tissue hypoxia.
Clinical Manifestations	General anemia symptoms.	Typically, don't develop until after age 40 in men and 50 in women. Fatigue, arthralgia, impotence, abdominal pain, & wgt loss. Iron accumulates in the liver later causing liver enlargement and cirrhosis. Liver, pancreas, heart, joints, and endocrine glands cause diabetes skin pigment changes, heart problems, arthritis, and testicular atrophy.	Splenomegaly and hepatomeg common. Increased clotting ri due to Hypervolemia and Hyperviscosity. Headache, ver dizziness, tinnitus, and visual Generalized itching (exacerba hot bath), paresthesias and erythromelalgia (painful burni redness of the hands and feet). HF, Intermittent claudication, thrombophlebitis. Most comm complication and major cause mortality is a stroke from thro Bleeding can be acute and cata
Diagnostic Studies	Hgb/Hct, MVC, Reticulocytes, Serum Iron, TIBC, Transferrin, Ferritin, Bilirubin, B12, Folate.	Total Iron exceeds 50g. High TIBC and serum ferritin. Testing for genetic mutations confirms diagnosis. MRI can measure liver and cardiac iron. Liver biopsy for amount of iron and degree of organ damage.	High Hgb, Hct, RBC Mass, Bo marrow examination, and pres mutations. Low EPO levels, H WBC count, Uric Acid, Cobal Levels.
Drug Therapy	General supportive care until the causative agent can be eliminated or made less injurious to the RBCs. Emergency treatment required- aggressive rehydration and electrolyte replacement. Corticosteroids and blood products or removing the spleen. Folate replacement.	Remove 500mL of blood each week until iron stores are depleted. Then blood removal becomes less and less to maintain appropriate levels. Iron-chelation therapy.	Reduce blood volume and visc bone marrow. Phlebotomy is a mainstay of treatment. Blood v to be reduced to reduce blood hydration therapy and aspirin. Myelosuppressive agents, Rux A-interferon-2b
Nursing Management	Administer medication, supportive treatments, blood products, blood testing.	Remove excess iron from the body and minimize symptoms. Manage problems from organ involvement. Initiate drug therapy and educations.	Assist with or perform phlebo Assess I/O and during hydrati therapy to avoid fluid overload deficit. Give medications and the patient and teach them abo side effects. Assess nutrition s encourage activity, assess for complications with each intera

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In order to receive full credit (2H class time) for this assignment, it must be completed in its entirety by the due date/time assigned. Any assignment not completed in its entirety by the due date and time will result in missed class time and must be completed by the end of the semester to pass the course.