Management of patient with hematological disorders

Learning Objectivities

On completion of this lecture the learner will be able to:

- Describe the process of hematopoiesis.
- Describe the significance of physical assessment and diagnostic test findings to the diagnosis of hematologic dysfunction.
- Identify therapies for blood disorders.

Key Terms

- Anticoagulant: an agent that prevents the clotting of blood.
  - Examples are EDTA, Citrate and Heparin
- Capillary: small blood vessel that connects arterioles and venules
- Hematoma: a subcutaneous mass of blood at a venipuncture site
- Hemoglobin: the oxygen carrying molecule of red blood cells
- Hemolysis: the breakdown of red blood cells, with the release of hemoglobin into the plasma or serum. Cannot use hemolyzed samples in lab tests

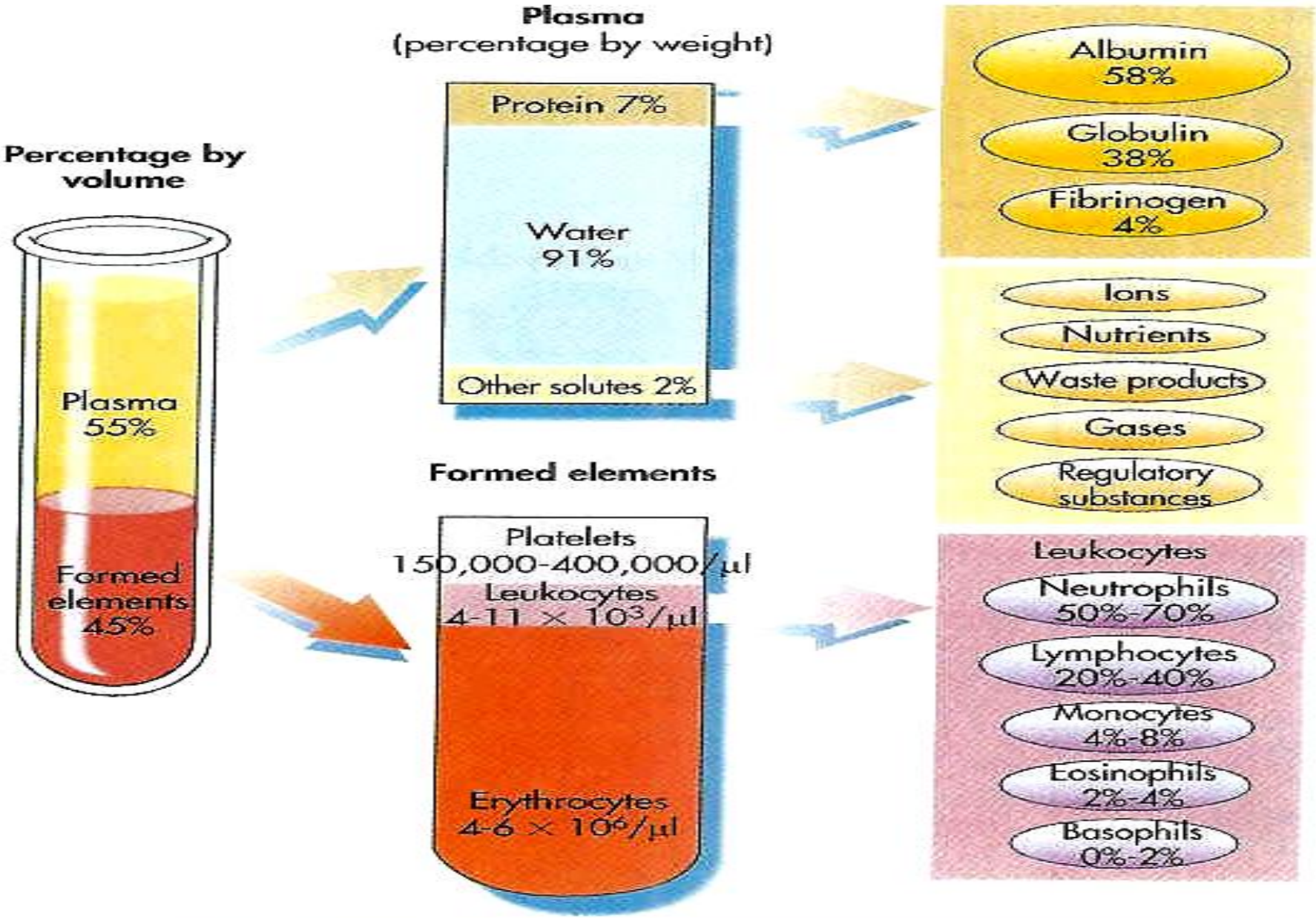
Anatomy and physiology

- Hematologic system consists of the blood and the sites where the blood is produced.
- Blood consists of plasma and blood cells.
- Because blood cells have a short lifespan, **hematopoiesis** (production od blood cells in the bone marrow) is needed.

Function of Blood

- Transporting fluids such as:
  - Nutrients from digestive tract
  - O_2 from lungs
  - Waste from cells
  - Hormones
- Aids in heat distribution
- Regulates acid-base balance

Blood Composition



Normal Ranges

- RBC: Female 3.6-5.0x$10^6 mm^3$
  Male 4.2-5.4x$10^6 mm^3$
- WBC: 4.5-10.5x$10^3 mm^3$ (African Americans is sltly lower 3.2 is still normal)
- HCT: female 36-48% male 42-52%
- Hgb: female 12-16 g/dL male 14-17.4 g/dL
- Platelets: 140-400x$10^3 mm^3$

Composition of Blood

- Plasma: liquid portion of blood w/out cells
  - Contains all of the following
    - Water Nutrients
    - Electrolytes Metabolic waste product
    - Hormones Vitamins and enzymes
    - Plasma proteins such as fibrinogen, albumin and globulin

Composition of Blood:

Erythrocytes

- Red blood cells are responsible for:
  - Transport of oxygen and nutrients
  - Removal of waste and CO_2 from the cells
  - Distribution of heat
- Hemoglobin:
  - The O_2 carrying potential.
  - Has 4 subunits, each contains a hem attached to a globin chain.
  - Iron is present in the hem, hem is bind to the oxygen

Composition of Blood:
Leukocytes

- WBC are responsible for:
  - Phagocytosis – to engulf and absorb waste material and harmful microorganisms in the blood stream and tissues
  - Synthesis of antibody molecules
  - Inflammation process
  - Production of heparin – component found in lung and liver tissue which have the ability to prevent clotting of blood.

Composition of Blood: Leukocytes (cont.)

- Types of Leukocytes
  - Granulocytes
    - Neutrophils
    - Eosinophils
    - Basophils
  - Agranulocytes
    - Lymphocytes
    - Monocytes

Composition of Blood: Thrombocytes

- Platelets – the smallest of the solid components of the blood
- Responsible for the clotting process
- Coagulation: term for clotting
- Embolism: a blood clot which is moving through the body

Blood Types

- Four Major Groups
  - A B AB O
- Blood types are inherited from your parents
- Antigen is present on the red blood cell; typing is done w/rbc
- Antibody is present in the plasma; antibody screening done on plasma

Blood Types

- O negative
  - Universal donor
  - It carries no antigen

- AB positive
  - Universal recipient
  - It carries no antibodies in the plasma

- 43% of population are O, 42% A, 12% B and 3% AB

ABO & Rh compatibility

- **Blood is classified according to the presence of these antigens:**
  - **Group A** contains **antigen A**
  - **Group AB** contains **both antigens**
  - **Group O** contains **neither antigen**

- **Blood plasma contains antibodies against the opposite antigen:**
- A person with Type **A blood** has antibodies against the **B antigen**
- A person with Type **AB blood** has **no antibodies** **(Universal Recipient)**

- A person with Type **O blood** has antibodies against the **A, B & AB** antigens **(Universal Donor)**

ABO & Rh compatibility

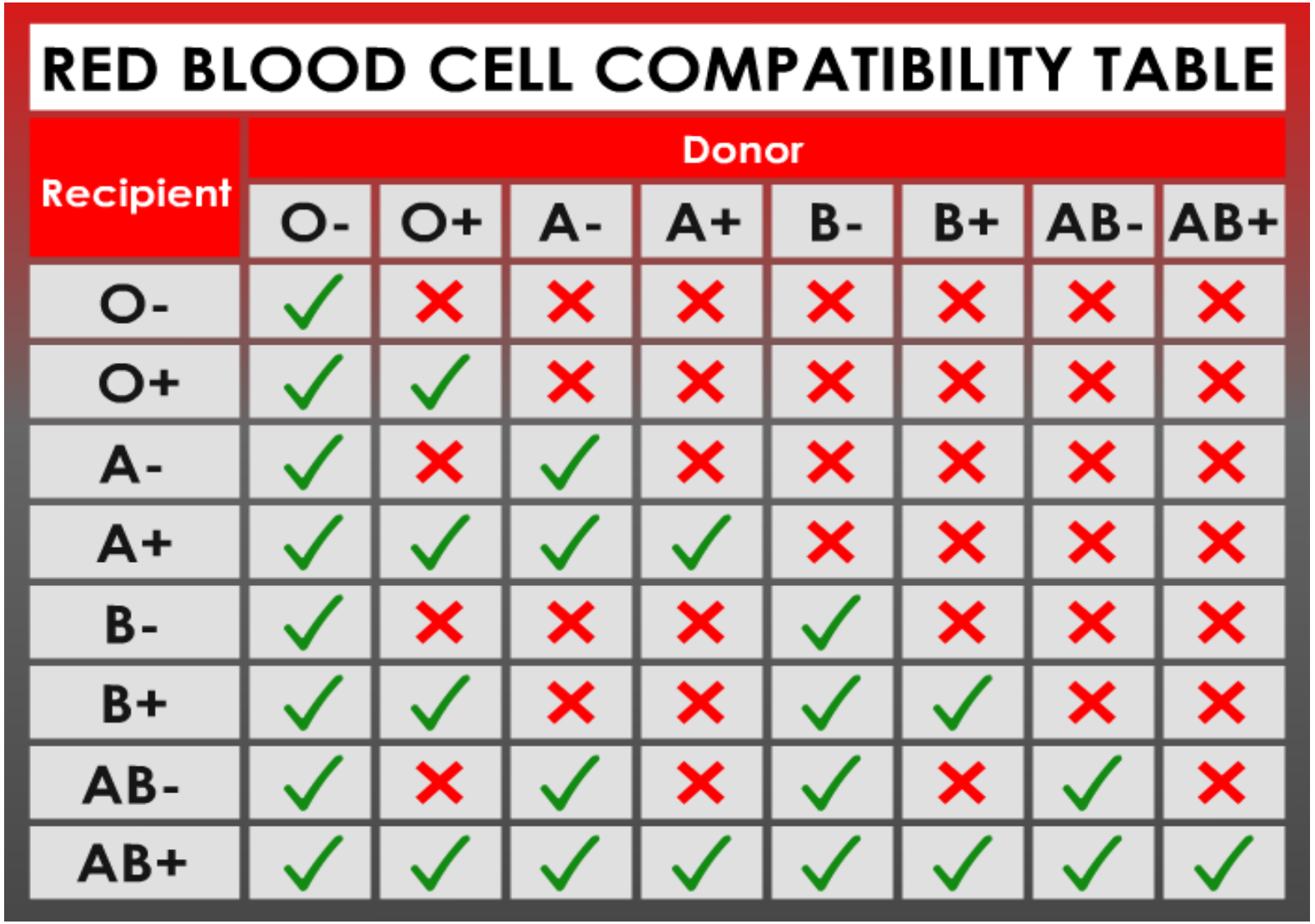
- People with Rhesus factors in their blood are classified as **"Rh positive"**
- People without the factors are classified as **"Rh negative"**
- Rh negative persons form antibodies against the Rh factor if they are exposed to Rh positive blood
- Conclusion:
  - Blood transfusion between incompatible groups causes an **immune response** against the cells carrying the antigen, resulting in **transfusion reaction**

Rh Factor

- found on the surface of rbc
- Rhesus factor: discovered in rhesus monkeys in 1937
- Can be phenotypically positive or negative
- Positive is dominant over negative
  - If positive is present, then you will express positive phenotype

	Recipient +	Recipient -
Donor +	OK	OK
Donor -	NO	OK

Blood Transfusion Compatibility Chart



RED BLOOD CELL COMPATIBILITY TABLE								
Recipient	Donor							
	O-	O+	A-	A+	B-	B+	AB-	AB+
O-	✓	✗	✗	✗	✗	✗	✗	✗
O+	✓	✓	✗	✗	✗	✗	✗	✗
A-	✓	✗	✓	✗	✗	✗	✗	✗
A+	✓	✓	✓	✓	✗	✗	✗	✗
B-	✓	✗	✗	✗	✓	✗	✗	✗
B+	✓	✓	✗	✗	✓	✓	✗	✗
AB-	✓	✗	✓	✗	✓	✗	✓	✗
AB+	✓	✓	✓	✓	✓	✓	✓	✓

Anemia

- Is a condition in which the hemoglobin concentration is lower than normal (low RBCs).
- As a result, the amount of oxygen delivered to the tissue is lower than normal.
- It is not a disease but a sign of underlying disorders.

Anemia

Ertyhropoiesis:

- Is the production Of RBC in the bone marrow
- When RBCs are low, the kidney releases erythropoietin to stimulate the bone marrow to produce more RBCs
- People who lve at high at high altitudes with lower atmospheric concentration, have more erythropoietin level in their bodies.
- Bone marrow required substances to produce RBCs which include: Iron, Vit B12 &6, flote, proteins, and others.
-

Classifications of Anemia

Type of Anemia	Laboratory Findings: CBC	Laboratory Findings: Other
Hypoproliferative (Resulting From Defective RBC Production)		
Iron deficiency	↓ MCV, ↓ reticulocytes	↓ Iron, % saturation, ferritin ↑ TIBC
Vitamin B_{12} deficiency (megaloblastic)	↑ MCV	↓ Vitamin B_{12}
Folate deficiency (megaloblastic)	↑ MCV	↓ Folate
Decreased erythropoietin production (e.g., from renal dysfunction)	Normal MCV	↓ Erythropoietin level ↑ Creatinine
Cancer/inflammation	Normal MCV	↑ Ferritin, % saturation ↓ Iron, TIBC ↓ Erythropoietin level (usually)
Bleeding (Resulting From RBC Loss)		
Bleeding from gastrointestinal tract, epistaxis (nosebleed), trauma, bleeding from genitourinary tract (e.g., menorrhagia)	↓ Hgb and Hct (*Note:* Hgb and Hct may be normal if measured soon after bleeding starts) ↓ MCV (normal MCV initially) ↑ Reticulocytes	↓ Iron, % saturation, ferritin (later)
Hemolytic (Resulting From RBC Destruction)		
Altered erythropoiesis (sickle cell anemia, thalassemia, other hemoglobinopathies)	↓ MCV ↑ Reticulocytes Fragmented RBCs (various shapes)	
Hypersplenism (hemolysis)	↑ MCV	
Drug-induced anemia	↑ Presence of spherocytes	
Autoimmune anemia	↑ Presence of spherocytes	
Mechanical heart valve–related anemia	Fragmented red cells	

CBC, complete blood count; RBC, red blood cell; ↓, decreased; MCV, mean corpuscular volume; %, percent; ↑, increased; TIBC, total iron-binding capacity; Hgb, hemoglobin; Hct, hematocrit.

Clinical Manifestations of Anemia

- Caused by the body's response to hypoxia
  - Mild (Hb 10 -14) no symptoms or minor changes
  - Moderate – (Hg 6 – 10) CV Changes: palpitations, dyspnea, diaphoresis
  - Severe – (Hg<6) multiple body system CV, Cerebral, Major Organs

Anemia
Clinical Manifestations

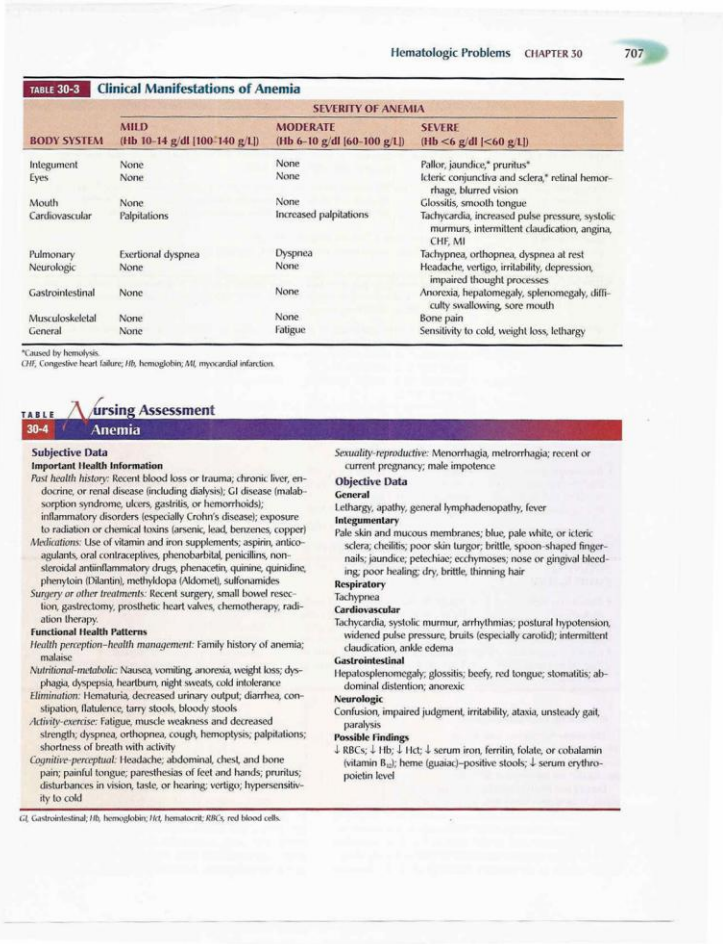


TABLE 30-3 Clinical Manifestations of Anemia

BODY SYSTEM	SEVERITY OF ANEMIA: MILD (Hb 10–14 g/dl [100–140 g/L])	MODERATE (Hb 6–10 g/dl [60–100 g/L])	SEVERE (Hb <6 g/dl [<60 g/L])
Integument	None	None	Pallor, jaundice,* pruritus*
Eyes	None	None	Icteric conjunctiva and sclera,* retinal hemorrhage, blurred vision
Mouth	None	None	Glossitis, smooth tongue
Cardiovascular	Palpitations	Increased palpitations	Tachycardia, increased pulse pressure, systolic murmurs, intermittent claudication, angina, CHF, MI
Pulmonary	Exertional dyspnea	Dyspnea	Tachypnea, orthopnea, dyspnea at rest
Neurologic	None	None	Headache, vertigo, irritability, depression, impaired thought processes
Gastrointestinal	None	None	Anorexia, hepatomegaly, splenomegaly, difficulty swallowing, sore mouth
Musculoskeletal	None	None	Bone pain
General	None	Fatigue	Sensitivity to cold, weight loss, lethargy

*Caused by hemolysis.
CHF, Congestive heart failure; *Hb,* hemoglobin; *MI,* myocardial infarction.

TABLE 30-4 Nursing Assessment: Anemia

Subjective Data

Important Health Information

Past health history: Recent blood loss or trauma; chronic liver, endocrine, or renal disease (including dialysis); GI disease (malabsorption syndrome, ulcers, gastritis, or hemorrhoids); inflammatory disorders (especially Crohn's disease); exposure to radiation or chemical toxins (arsenic, lead, benzenes, copper)

Medications: Use of vitamin and iron supplements; aspirin, anticoagulants, oral contraceptives, phenobarbital, penicillins, nonsteroidal antiinflammatory drugs, phenacetin, quinine, quinidine, phenytoin (Dilantin), methyldopa (Aldomet), sulfonamides

Surgery or other treatments: Recent surgery, small bowel resection, gastrectomy, prosthetic heart valves, chemotherapy, radiation therapy.

Functional Health Patterns

Health perception–health management: Family history of anemia; malaise

Nutritional-metabolic: Nausea, vomiting, anorexia, weight loss; dysphagia, dyspepsia, heartburn, night sweats, cold intolerance

Elimination: Hematuria, decreased urinary output; diarrhea, constipation, flatulence, tarry stools, bloody stools

Activity-exercise: Fatigue, muscle weakness and decreased strength; dyspnea, orthopnea, cough, hemoptysis; palpitations; shortness of breath with activity

Cognitive-perceptual: Headache; abdominal, chest, and bone pain; painful tongue; paresthesias of feet and hands; pruritus; disturbances in vision, taste, or hearing; vertigo; hypersensitivity to cold

Sexuality-reproductive: Menorrhagia, metrorrhagia; recent or current pregnancy; male impotence

Objective Data

General

Lethargy, apathy, general lymphadenopathy, fever

Integumentary

Pale skin and mucous membranes; blue, pale white, or icteric sclera; cheilitis; poor skin turgor; brittle, spoon-shaped fingernails; jaundice; petechiae; ecchymoses; nose or gingival bleeding; poor healing; dry, brittle, thinning hair

Respiratory

Tachypnea

Cardiovascular

Tachycardia, systolic murmur, arrhythmias; postural hypotension, widened pulse pressure, bruits (especially carotid); intermittent claudication, ankle edema

Gastrointestinal

Hepatosplenomegaly; glossitis; beefy, red tongue; stomatitis; abdominal distention; anorexic

Neurologic

Confusion, impaired judgment, irritability, ataxia, unsteady gait, paralysis

Possible Findings

↓ RBCs; ↓ Hb; ↓ Hct; ↓ serum iron, ferritin, folate, or cobalamin (vitamin B_{12}); heme (guaiac)–positive stools; ↓ serum erythropoietin level

GI, Gastrointestinal; *Hb,* hemoglobin; *Hct,* hematocrit; *RBCs,* red blood cells.

Iron-Deficiency Anemia

Etiology: Inadequate dietary intake, malabsorption, blood loss, or hemolysis

Clinical Manifestations:

- **Pallor**
- **Glossitis – inflammation of the tongue**
- **Cheilitis – inflammation of the lips**
- **Headache, paresthesia, burning sensation of the tongue**

Diagnostic Studies: Lab Studies, Endoscopy to identify GI bleed

Treatment:

- **Oral Iron replacement (Iron absorbed best in duodenum)**
- **Ferrous sulfate – take about one hour prior to meal**

Nursing Management – Diet & Medication Instruction

Thalassemia

Genetic disorder of inadequate production of normal hemoglobin

- Hemolysis occurs
- Abnormal Hb synthesis
- Ethnic groups of Mediterranean Sea & near equatorial regions of Asia and Africa

Clinical Manifestation:

- mild – moderate anemia with hypochromia (pale cells) or microcytosis (small cells)
- Minor: one thalassemic gene – mild
- Major: two thalassemic genes – severe – physical & mental growth retarded - cardiac failure is fatal

Medical Management:

- Medication: **Chelation Therapy** IV deferoxamine (Desferal) – iron binding agent to reduce iron overload
- Transfusions to maintain Hg >10g/dl

Nursing Management: Supportive

Megaloblastic Anemias

Caused by impaired DNA synthesis & characterized

by the presence of large RBCs

Causes: Pernicious anemia, nutritional deficiency; heredity enzyme defect

Clinical Manifestations: GI—sore tongue, anorexia, N&V, abdominal pain; muscle weakness, paresthesias of feet and hands; confusion

Diagnostic Testing: Serum cobalamin levels; gastroscopy; Schilling Test – assesses parietal cell function

Medical Management: Parenteral administration of cobalamin – daily for 2 weeks, then weekly until >HCT, then monthly for life; intranasal form

Nursing Management: Health Promotion; protection from sensory injury—burns, trauma; pt compliance with replacement therapy

Anemia of Chronic Disease

- **Associated** with underproduction of RBCs and decreased RBC survival
- **Causes**: Renal failure; advanced liver cirrhosis; chronic inflammation; malignancy; immunosuppression
- **Medical Management:**
  - Correct underlying disorder
  - Erythropoietin Therapy – Epogen, Procrit
- **Nursing Management:** Care of the debilitated patient – dietary & medication compliance

Anemia Caused by Blood Loss

- **Acute Blood Loss**
  - Hemorrhage
- **Chronic Blood Loss**
  - Body maintains its blood volume by slowly increasing plasma volume < RBCs

- **Clinical Manifestations:**
  - Range from fatigue with melena to orthostatic BP changes to shock

- **Medical Management:**
  - Treat underlying cause –
  - Blood replacement – packed RBCs
  - Supplemental Iron

Sickle Cell Disease

- Group of inherited autosomal recessive disorders characterized by the presence of abnormal Hgb in the erythrocyte
  - Causes the erythrocyte to stiffen & elongate
  - Sickle shape in response to lack of oxygen
- **Types:**
  - Sickle Cell Anemia: most severe – inherited homozygous for hemoglobin S (HbSS) from both parents
  - Sickle Cell Trait: mild - inherited from one parent + one normal

- **Sickling Episodes:**
  - Hypoxemia – triggered by stress, surgery, blood loss, viral or bacterial infection*(most common), dehydration, acidosis
  - Hemolyzed in the spleen
  - Initially reversible – then becomes irreversible due to chronic sickling

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Patient with Anemia

EXPECTED PATIENT OUTCOMES	NURSING INTERVENTIONS and *RATIONALES*
NURSING DIAGNOSIS	**Activity intolerance** *related to* weakness and malaise *as manifested by* difficulty in tolerating increased activity (e.g., increased pulse, respiration).
▪ Participation in activities of daily living (e.g., bathing, dressing, grooming, feeding) to greatest extent possible ▪ Vital signs within acceptable range	▪ Plan care to alternate periods of rest and activity *to provide activity without tiring the patient.* ▪ Strive for a 1:3 rest/activity ratio; assist patient with activities of daily living as needed. ▪ Limit visitors, phone calls, noise, and interruptions by hospital staff *to reduce demands placed on patient.* ▪ Monitor vital signs *to evaluate activity tolerance.* ▪ Monitor hematocrit and hemoglobin *as a guide to planning activities.*
NURSING DIAGNOSIS	**Imbalanced nutrition: less than body requirements** *related to* poor nutritional intake, anorexia, and treatment *as manifested by* weight loss, low serum albumin, decreased iron levels, vitamin deficiencies, below usual body weight.
▪ Maintenance of body weight, then gradual increase to within range of ideal body weight ▪ Hematocrit, hemoglobin, and serum albumin within normal ranges	▪ Teach patient about foods high in protein, iron, calories, and other nutrients *to increase intake of essential nutrients needed for hematopoiesis* (see Table 30-5). ▪ With input from patient, establish range of optimal weight outcomes and dietary plan *to involve patient and increase compliance.* ▪ Teach and monitor use of a food diary *to increase patient's awareness of actual intake and increase intake.* ▪ Suggest eating small, frequent meals with snacks throughout the day.
NURSING DIAGNOSIS	**Ineffective therapeutic regimen management** *related to* lack of knowledge about appropriate nutrition and medication regimen *as manifested by* questioning about lifestyle adjustments, diet, medication prescriptions.
▪ Knowledge about lifestyle changes, nutrition, and medication regimens	▪ Review and teach patient about nutrition and medication information *to promote compliance.* ▪ Teach about and monitor response to supplemental drugs that aid in red blood cell production *because it is often difficult to correct anemia by diet alone.* ▪ Suggest follow-up resources *to help patient maintain gains and adjustments throughout recovery.*

COLLABORATIVE PROBLEM

NURSING GOALS	NURSING INTERVENTIONS and *RATIONALES*
POTENTIAL COMPLICATION	**Hypoxemia** *related to* decreased hemoglobin.
▪ Monitor for signs of hypoxemia ▪ Report deviations from acceptable parameter ▪ Carry out appropriate medical and nursing interventions	▪ Assess for manifestations of hypoxemia such as dyspnea, decrease in O_2 saturation, increase in $PaCO_2$, cyanosis *to initiate early intervention.* ▪ Administer O_2 as ordered *to saturate all available hemoglobin.* ▪ Transfuse with blood products as ordered *to increase red blood cells.* ▪ Monitor hemoglobin *to determine severity of anemia and response to treatment.* ▪ Teach effective breathing exercises and relaxation techniques *to relieve dyspnea and to promote maximum thoracic excursion.*

Leukemia

Definition

It is a group of malignant disorder, affecting the blood and blood –forming tissue of the bone marrow lymph system and spleen.

A etiology

- Combination of predisposing factors including genetic and environmental influences.
- Chronic exposure to chemical such as benzene
- Radiation exposure.
- Cytotoxic therapy of breast, lung and testicular cancer.

Classification of leukaemia

1. **1. Acute lymphatic leukaemia (ALL)**

   Usually occurs before 14 years of age peak incidence is between 2-9 years of age, older adult

Pathophysiology

It arising from a single lymphoid stem cell, with impaired maturation and accumulation of the malignant cells in the bone marrow.

Acute lymphatic leukaemia Cont.

Signs and symptoms

Anaemia, bleeding, lymphadenopathy, infection

Clinical manifestation	Clinical manifestation
▪Fever ▪Pallor ▪Bleeding ▪Anorexia ▪Fatigue ▪Weakness ▪Bone, joint and abdominal pain ▪Increase intracranial press.	▪Generalized lymphadenopathy ▪Infection of respiratory tract ▪Anaemia and bleeding of mucus membrane ▪Ecchymoses ▪Weight loss ▪Hepatomegaly ▪Mouth sore

Acute lymphatic leukaemia Cont.

Management

Diagnosis

- Low RBCs count, Hb, Hct, low platelet count , low normal or high WBC count.
- Blood smear show immature lymph blasts.

Treatment

Chemotherapeutic agent, it involve three phases

1. **Induction:** Using vincristine and prednisone.
2. **Consolidation:** Using modified course of intensive therapy to eradicate any remaining.
3. **Maintenance**

Acute lymphatic leukaemia Cont.

Treatment Cont.

- Prophylactic treatment of the CNS , intrathecal administration and /or craniospinal radiation with eradicate leukemic cells.

- Eat diet that contains high in protein, fibres and fluids.

Acute lymphatic leukaemia Cont.

Treatment Cont.

- Avoid infection (hand washing, avoid crowds),injury
- Take measure to decrease nausea and to promote appetite, smoking and spicy and hot foods.
- Maintain oral hygiene.

Acute Myelogenous Leukaemia (AML)

It occurs at any age but occurs most often at adolescence and after age of 55

Pathophysiology

Characterized by the development of immature myeloblasts in the bone marrow.

Clinical manifestation

Similar to ALL plus sternal tenderness.

Management

Diagnosis

Low RBC, Hb, Hct, low platelet count, low to high WBC count with myeloblasts.

Acute Myelogenous Leukaemia (AML) Cont.

Treatment

- Use of cytarabine, 6-thioquanine, and doxorubic
- The same care of client as All, plus give adequate amounts of fluids(2000 to 3000 ml per day.)
- Instruct client about medication, effects, side effects and nursing measures

Chronic lymphocytic Leukaemia (CLL)

The incidence of CLincreases with age and is rare under the age of 35.It is common in men.

Pathophysiology

- It is characterized by proliferation of small, abnormal , mature B lymphocytes, often leading to decreased synthesis of immunoglobulin and depressed antibody response.
- The number of mature lymphocytes in peripheral blood smear and bone marrow are greatly increased

Chronic lymphocytic Leukaemia (CLL) Cont

Clinical Manifestation

Usually there is no symptoms.

Chronic fatigue , weakness , anorexia, splenomegaly , lymphadenopathy, hepatomegaly.

Signs and Symptoms

- Pruritic vesicular skin lesions .
- Anaemia
- Thrombocytopenia.
- The WBC count is elevated to a level between 20,000 to 100,000.
- Increase blood viscosity and clotting episode.

Chronic lymphocytic Leukaemia (CLL) Cont

Management

I. Persons are treated only when symptoms, particular anaemia , thrombocytopenia , enlarged lymph nodes and spleen appear.

I. Chemotherapy agents such as chlorambucil , and the glucocorticoids.

I. Client and family education is that describe for AML.

Chronic Myelogenous Leukaemia(CML)

Occurs between 25-60 years of age. Peak 45 year

It is caused by benzene exposure and high doses of radiation.

Clinical Manifestation

- There is no symptoms in disease. The classic symptoms of chronic types of leukaemia, include:
- Fatigue, weakness, fever, sternal tenderness.
- Weight loss, joint & bone pain.

Chronic Myelogenous Leukaemia(CML) Cont.

Clinical Manifestation Cont.

- Massive splenomegaly and increase in sweating.
- The accelerated phase of disease(blostic phase) is characterized by increasing number of granulocytes in the peripheral blood.
- There is a corresponding anaemia and thrombocytopenia.

Questions?