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DISORDERS OF RBC: ANEMIA

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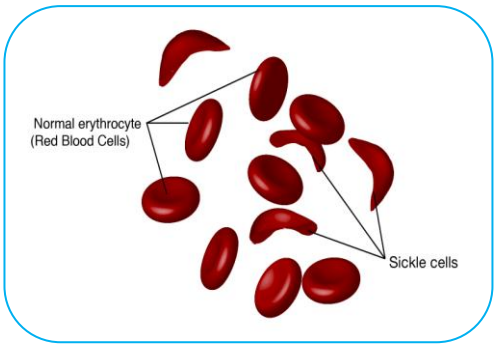
PREFACE:

Anemia is a blood disorder, characterized by the reduction in:
1. Red blood cell (RBC) count
2. Hemoglobin content
3. Packed cell volume (PVC)



Anemic condition generally precipitates because of:

1. Decreased production of RBC
2. Increased destruction of RBC
3. Excess loss of blood from the body

These changes in blood are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins.

CLASSIFICATION OF ANEMIA

Anemia is classified based on two criteria;
1. Etiology
2. Morphology

ETIOLOGICAL CLASSIFICATION

On the basis of etiology (study of cause or origin), anemia is divided into five types;
1. Anemia of chronic disease
2. Aplastic anemia
3. Hemorrhagic anemia
4. Hemolytic anemia
5. Nutrition deficiency anemia

1. Anemia of Chronic Diseases: Anemia of chronic diseases is the second most common type of anemia (next to iron deficiency anemia). Disturbance in iron metabolism or resistance to erythropoietin action leads to short lifespan of RBCs. Temporally, anemia sets in only after few months after disease

manifestation. RBCS are however normal in size (normocytic) and color (normochromic).

Common causes of anemia of chronic diseases:

i. Non-infectious inflammatory diseases such as rheumatoid arthritis (chronic inflammatory autoimmune disorder affecting joints).
ii. Infections such as tuberculosis and lung abscesses
iii. Chronic renal failure, in which the erythropoietin secretion decreases (because erythropoietin is necessary for the stimulation of bone marrow to produce RBCs, its deficiency causes anemia).
iv. Neoplastic disorders featuring abnormal cell growth such as Hodgkin's disease (malignancy involving lymphocytes) and cancer of lung and breast.

RBCs are generally normocytic and normochromic in this type of anemia. However, in progressive disease associated with iron deficiency the cells become microcytic and hypochromic.

2. Aplastic anemia: Aplastic anemia is a disorder of red bone marrow. In this case, red bon e marrow is reduced and replaced by fatty tissues. Disorders of Bone marrow occurs due to;

i. Frequent exposure to radiation caused by X-rays or gamma rays
ii. Presence of bacterial toxins, quinine, gold salts, benzene, radium, etc
iii. Tuberculosis
iv. Viral infections like hepatitis and HIV infections

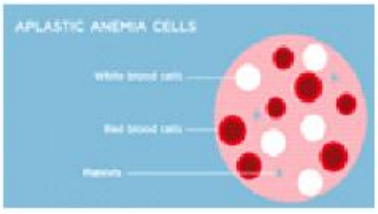


Figure 1: Aplastic anemia normal RBCs with normal size

In aplastic anemia, RBCs are however normocytic and normochromic (Fig. 1)

3. Hemolytic Anemia: Hemolysis means destruction of RBCs. In hemolytic anemia excessive hemolysis of RBCs is noted, and it is not compensated by increased RBC production. Hemolytic anemia is of two types depending upon the causative factor:

A. Extrinsic hemolytic anemia
B. Intrinsic hemolytic anemia

A. Extrinsic hemolytic anemia: It is the type of anemia caused because of RBCs destruction by external factors including antibodies, chemicals and drugs. Extrinsic hemolytic anemia is also termed 'autoimmune hemolytic anemia'.

Common causes of extrinsic hemolytic anemia are;

i. Liver failure
ii. Renal disorder
iii. Hypersplenism
iv. Burns.
v. Hepatitis, malaria and septicemic infections
vi. Medications including penicillin, antimalarial and Sulfa drugs.
vii. Poisoning by chemical substances like lead, coal and tar
viii. Presence of isoagglutinins, ex- anti-Rh
ix. Autoimmune disorders such as rheumatoid Arthritis and ulcerative colitis.

B. Intrinsic hemolytic anemia: It is caused due to destruction of RBCs because of inherited defects in RBCs. Unhealthy RBCs that live and die soon are produced. Intrinsic hemolytic anemia is often inherited and it includes sickle cell anemia and thalassemia. Because of the abnormal shape of RBCs in sickle cell anemia and thalassemia, the RBCs become more fragile and are susceptible to hemolysis.

a. Sickle cell anemia: Sickle cell anemia or Hemoglobin SS disease is an inherited disorder of RBCs, which is characterized by sickle-shaped red blood cells. It is common in people of African origin. Sickle cell anemia is due to the presence of abnormal hemoglobin in RBCs called hemoglobin S (sickle cell hemoglobin). In this, alpha chains are normal and beta chains are abnormal leading to polymerization of hemoglobin S molecules into long chains that precipitate inside the cells. Because of this, the RBCs attain sickle (crescent) shape and become more fragile leading to hemolysis. Sickle cell anemia is a recessive mutation genetic disorder, where a person inherits two abnormal S gene mutations (one from each parent). In children, hemolyzed sickle cells aggregate and block the blood vessels, leading to infarction (stoppage of blood supply), seen especially in small bones. These infarcted small bones in hand and foot results in varying lengths of the digits, which is referred to as hand and foot syndrome. Jaundice also occurs in these children.

B.Thalassemia: Thalassemia is an inherited disorder, characterized by abnormal hemoglobin that has less oxygen carrying capacity (Fig. 2). Thalassemia is also known as Cooley's or Mediterranean anemia. It is more common in Thailand and to some extent in Mediterranean countries.
Thalassemia is of two types;

i. A (alpha) thalassemia
ii. B (beta) -thalassemia

Figure 2: Malformed RBC in Thalassemia

i. A-Thalassemia: A- thalassemia occurs infancy or fetal life. In this alpha chains are less, absent or abnormal. In adults, beta chains are in excess and in children, y-chains are in excess. A-thalassemia leads to defective erythropoiesis and hemolysis. Consequently, infants may be stillborn or die immediately after birth.

ii. B-Thalassemia: In B-thalassemia, beta -chains are less in number, absent or abnormal with an excess of alpha chains. The alpha chains precipitate defective erythropoiesis and hemolysis. The B-thalassemia is very common among these two conditions. In normal hemoglobin, number of alpha and beta polypeptide chains is equal. In thalassemia, the production of these chains become imbalanced because of defective synthesis of one or other chains of the globin gene, which leads to decreased production of alpha or beta globin chains and altered balance of the two globin chains. This causes the precipitation of the polypeptide chains in the immature RBCs, leading to disturbance in erythropoiesis. The precipitation also occurs in mature red cells, resulting in hemolysis.

4. Nutrition Deficiency Anemia: Deficiency of a nutritive substance required for erythropoiesis leads to anemia. The substances which are necessary for erythropoiesis are iron, proteins and vitamins like vitamin C, B12 and folic acid. The types of nutrition deficiency anemia are:

A. Iron deficiency anemia: Iron deficiency anemia results from inadequacy of iron for hemoglobin synthesis. It is one of most common types seen in anemic patients with characteristic appearance of microcytic and hypochromic RBCs.

Iron deficiency anemia results from;
i. Loss of blood
ii. Decreased iron intake
iii. Poor absorption of iron from intestine
iv. Increased demand for iron during periods of rapid growth and pregnancy.

Iron deficiency anemia patients may also have brittle and spoon shaped nails (koilonychias), brittle hair, atrophy of papilla in tongue and dysphagia (difficulty in swallowing).
B. Protein deficiency anemia: Protein deficiency leads to decreased synthesis of hemoglobin, which leads to generation of pale (hypochromic) and large sized (macrocytic) RBCs.

C. Pernicious anemia or Addison's anemia: Pernicious anemia results because of deficiency of vitamin B12. It results due to autoimmune destruction of parietal cells and gastric mucosa, resulting in decreased production of intrinsic factor and poor absorption of vitamin B12 that is the maturation factor for RBC. RBCs are larger and immature with near normal or slightly low hemoglobin level. So, cells are macrocytic and normochromic or slightly hypochromic.

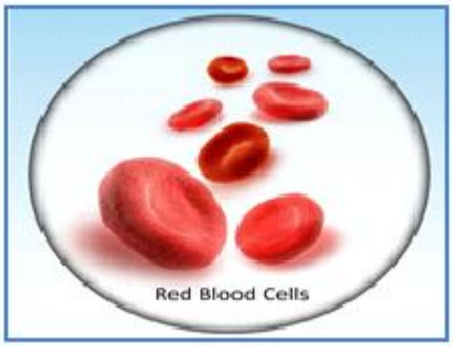


Figure 3: **Pernicious Anemia**: Larger RBCs with normal or slightly low levels of hemoglobin

Before discovering the cause of this anemia, it was very difficult to treat the patients and the disease was considered to be fatal. So, it was called pernicious anemia. Pernicious anemia is common in old age and it is more common in females than in males. It is associated with other autoimmune disorders of thyroid gland and adrenal gland. Characteristic features of this type of anemia are lemon yellow color of skin (due to anemic paleness and mild jaundice) and red sore tongue. Neurological disorders such as paresthesia featuring numbness, tingling, burning, etc.), progressive weakness and ataxia (muscular incoordination) may also manifest.

D. Megaloblastic anemia: Megaloblastic anemia results due to the deficiency of another maturation factor called folic acid. Here, the RBCs are not matured. The DNA synthesis is also defective, so the nucleus remains immature. Therefore, RBCs are megaloblastic and hypochromic. However, some characteristic features of pernicious anemia appear in megaloblastic anemia also.

MORPHOLOGICAL CLASSIFICATION:

Morphological classification is based on the size and color of RBC. Size of RBC is determined by mean corpuscular volume (MCV). RBCs Color is determined by mean corpuscular hemoglobin concentration (MCHC). By this method, the anemia is classified into four types

i. Normocytic Normochromic Anemia: Size (MCV) and color (MCHC) of RBCs are normal. But the number of RBC is less.
ii. Macrocytic Normochromic Anemia: Although RBC count declines, RBCs are larger in size and display normal color.
iii. Macrocytic Hypochromic Anemia: RBCs are larger in size, but look pale because of less MCHC.
iv. Microcytic Hypochromic Anemia: RBCs are smaller in size and pale in color.

SIGNS AND SYMPTOMS OF ANEMIA:

i. Skin: Color of the skin and mucous membranes becomes pale. Paleness of buccal and pharyngeal mucous membrane, conjunctivae, lips, ear lobes, palm and nail bed. Skin becomes thin, elastic and dry. Thinning, loss and early grayness of hair occurs. The nails become brittle and easily breakable.
ii. Cardiovascular system: Heart rate increases (tachycardia) and cardiac output increases. Heart is dilated and cardiac murmurs are produced. The velocity of blood flow is increased.
iii. Respiration: Increase in rate and force of respiration is observed, which frequently leads to breathlessness and dyspnea (difficulty in breathing). Oxygen-hemoglobin dissociation curve is shifted to right.
iv. Digestion: Digestive disorders including anorexia, nausea, vomiting, abdominal pain and constipation

are common. Atrophy of tounge papillae is more common in pernecious anemia. In aplastic anemia, necrotic lesions appear in mouth and pharynx.
v. Metabolism: Basal metabolic rate increases in severe anemia.
vi. Kidney: Disturbed renal function and albuminuria is common.
vii. Reproductive system: The menstrual cycle is frequently disturbed in females. There may be menorrhagia, oligomenorrhea or amenorrhea.
viii. Neuromuscular system: These symptoms include increased sensitivity to cold, headache, lack of concentration, restlessness, irritability, drowsiness, dizziness or vertigo and fainting. Patient feels muscular weakness, lethargic and fatigued quite often and quite easily.

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