McCance: Pathophysiology, 6th Edition

Chapter 26: Alterations of Erythrocyte Function

Key Points – Print

SUMMARY REVIEW

Anemia

1. Anemia is defined as a reduction in the number or volume of circulating RBCs or a decrease in hemoglobin. Polycythemias are excessive levels of RBCs or volume.
2. Anemias can be classified according to (1) erythrocyte size or concentration of hemoglobin or (2) their cause.
3. Clinical manifestations of anemia may be demonstrated in all organs and tissues (tissue hypoxia) throughout the body. Decreased oxygen delivery to tissues causes fatigue, dyspnea, syncope, angina, compensatory tachycardia, and organ dysfunction.
4. Macrocytic-normochromic, or megaloblastic-normochromic, anemias are characterized by larger than normal RBCs with normal levels of hemoglobin. They most commonly are caused by deficiency of vitamin B_{12} (PA) or folate.
5. PA results from inadequate vitamin B_{12} absorption because of autoantibodies against the B_{12} transporter IF. Folate deficiency anemia is caused by inadequate dietary intake of folate. Both anemias respond to replacement therapy.
6. Microcytic-hypochromic anemias are characterized by abnormally small RBCs with insufficient hemoglobin content. This disorder results from disorders of (1) iron metabolism (IDA), (2) porphyrin and heme synthesis (SAs), or globin synthesis (thalassemia).
7. IDA is the most common type of anemia worldwide. It usually develops slowly, with gradual insidious onset of symptoms. Fatigue, weakness, dyspnea, alteration of various epithelial tissues, and vague neuromuscular complaints result.
8. IDA is usually a result of blood loss or poor nutritional intake. Individuals at highest risk for developing IDA older adults, women, infants, and those living in poverty. Anemia is also recognized as part of the nonspecific acute phase response to any type of inflammation. Once the source of blood loss is identified and corrected, oral iron replacement therapy can be initiated.
9. SA result from defects in mitochondrial metabolism leading to ineffective iron uptake and dysfunctional heme synthesis. The characteristic cell in the bone marrow, a ringed sideroblast, is an erythroblast containing iron granules arranged around the nucleus. SAs may be hereditary or acquired, and treatment varies depending on the cause.
10. Normocytic-normochromic anemias are characterized by insufficient numbers of normal erythrocytes. Included in this category are aplastic, posthemorrhagic, and hemolytic anemias and ACD.

11. AA is a critical condition characterized by a reduction or absence of all three blood cell types (pancytopenia). Unless the cause is determined, bone marrow aplasia results in death.
12. Acute blood loss from hemorrhage results in posthemorrhagic anemia with the severity depending on the amount of hemorrhage. Restoration of blood volume by plasma expanders or transfusions may diminish subjective symptoms of anemia. Hemoglobin restoration may take 6 to 8 weeks.
13. Hemolytic anemia is a result of excessive destruction of erythrocytes and may be acquired or hereditary. Of the acquired forms, autoimmune reaction (immunohemolytic) and drug-induced hemolysis are the most common.
14. AIHAs include (1) warm reactive antibody type, (2) cold agglutinin type, and (3) cold hemolysin type (paroxysmal cold hemoglobinuria).
15. ACD results from decreased erythropoiesis secondary to chronic diseases. The anemia is mild to moderate and one of the most common conditions encountered in medicine.
16. Mechanisms associated with ACD include (1) decreased erythrocyte life span, (2) reduced production of erythropoietin, (3) ineffective bone marrow response to erythropoietin, and (4) iron sequestration in macrophages. In particular, the proinflammataory cytokine IL-6 increases hepatocyte release of hepcidin, which suppresses ferroportin transport of iron out of macrophages.

Myeloproliferative RBC Disorders (Polycythemia)

1. Polycythemia vera is a myeloproliferative disorder characterized by excessive proliferation of erythrocyte precursors in the bone marrow. Signs and symptoms result directly from increased blood volume and viscosity and a predisposition to thrombosis.
2. Therapeutic phlebotomy to remove excessive blood volume and the use of hydroxyurea have been helpful in decreasing the excessive RBC population.