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Types of anemia and Physiological anemia of infancy – A Review

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Abstract

Iron deficiency is the most common nutritional disorder in the world. It is estimated that 30% of the world's population suffers from iron deficiency anemia, and most of them live in developing countries. There is about 0.5 grams of iron in the body of a baby, while the body's iron content is about 5 grams. This difference in iron levels from birth to adolescence means that an average of 0.8 mg of iron should be absorbed per day in the early 15 years of life. On the other hand, iron is also needed to compensate for the loss of iron due to natural degradation. Therefore, about 1 mg of iron should be absorbed daily to maintain the positive balance of iron in childhood.In newborn babies, the highest levels of iron are found in hemoglobin. During the first 2 to 3 months of life, when high concentrations of hemoglobin are reduced in an infant, iron is significantly altered and stored in the body. In infants, improved reserves are usually sufficient for hematopoiesis during the first 6 to 9 months of life.The absorption of iron in infants who fed from breast milk is 2 to 3 times higher than those infants feeding on cow's milk.

Keywords: anemia, Physiological anemia, infancy

Introduction

The functional classification of anemia consists of three major groups which are like the followings (1-3):

1- Bone marrow production (hypo-proliferative anemia), which includes iron deficiency anemia, is a form of anemia associated with chronic, renal and inflammatory diseases.

2. Disorders of evolution of red blood cells and ineffective erythropoiesis that develop in various diseases including thalassemia, Sidrobethemicanemia, and Megalobesticusanemia

3. Reduced survival of red blood cells (hemolysis and loss of blood): Intravascular hemolysis, hemoglobinopathy, loss of blood and etc.

Anemia caused by reduced production is the most common forms of anemia with acute and severe inflammation being the most common cause of this disorder. Anemia caused by inflammation like iron deficiency is partly linked to iron metabolism problems. Anemia features associated with kidney disease, inflammation, cancer, and metabolic abnormality are erythropoietin abnormal responses to anemia.

Iron deficiency anemia

Iron deficiency is the most common nutritional disorder in the world. It is estimated that 30% of the world's population suffers from iron deficiency anemia, and most of them live in developing countries (4). There is about 0.5 grams of iron in the body of a

baby, while the body's iron content is about 5 grams. This difference in iron levels from birth to adolescence means that an average of 0.8 mg of iron should be absorbed per day in the early 15 years of life. On the other hand, iron is also needed to compensate for the loss of iron due to natural degradation (5). Therefore, about 1 mg of iron should be absorbed daily to maintain the positive balance of iron in childhood. Since usually less than 10% of iron is absorbed into food, it is urgently required to maintain iron levels of 8 to 10 mg per day, due to the fact that the infant's growth rate is high and that the amount of iron in the cow's milk or breast milk is about one milligram, and this endangers keeping the body's iron. The absorption of iron in infants who fed from breast milk is 2 to 3 times higher than those infants feeding on cow's milk (6-8).

Etiology

In newborn babies, the highest levels of iron are found in hemoglobin. During the first 2 to 3 months of life, when high concentrations of hemoglobin are reduced in an infant, iron is significantly altered and stored in the body. In infants, improved reserves are usually sufficient for hematopoiesis during the first 6 to 9 months of life (9).

In infants, anemia, which is caused only by dietary iron deficiency, usually occurs between the ages of 9 and 24 months; afterwards, it is relatively uncommon. A diet found commonly in infants with iron deficiency anemia in developed countries is a form rich with cow's milk (low iron content, gastrointestinal bleeding due to leukocyte from milk protein) which often causesobesity in a child; malnutrition is often the cause of iron deficiency throughout the world (10).
In progressive iron deficiency, anemia occurs in a series of chemical and blood events. Clinical diagnosis of iron deficiency anemia is not so difficult. At first, tissue iron stores are empty and there occurs a significant decrease in tissue stores as a result of the drop in serum ferritin levels, which is a reserve iron protein. In the absence of inflammatory disease, ferritin provides an accurate estimation of iron stores, lowering serum iron levels, increasing the serum iron binding capacity (serum transferrin) and transferring saturation to less than normal levels (11). When iron storage decreases, iron availability is limited to combining with protoporphyrin to form hemoglobin. Free protoporphyrin accumulates in erythropoietin, and as a result, hemoglobin production is impaired. It should be noted that with the development of iron deficiency, iron deficiency anemia occurs. Red blood cells gets smaller and smaller in case of limited access to hemoglobin (12).

Physiological anemia of infancy

Newborn infants are more likely to have higher hemoglobin and hematocrit with larger red blood cells than older children and the adults. However, in the first week of life, progressive reduction of hemoglobin levels begins and lasts for 6 to 4 weeks. The result of this reduction is called the physiological anemia of infancy, a disorder in which various factors seem to be involved (13).

Postpartum respiration begins with a large amount of oxygen for transfer to hemoglobin, followed by an oxygen saturation of 50% to 90% or more, in addition to naturally produced embryonic hemoglobin after birth (14).Hemoglobin changes in adults is more willing to bind with oxygen in comparison with infant hemoglobin and, on the other hand, adult hemoglobin has a higher ability to oxygenate the tissues. Increasing blood oxygen levels and improving oxygen supply to the tissues leads to decreased production of erythropoietin and inhibition of hematopoiesis (15). In the absence of hematopoiesis, due to the fact that worn red blood cells naturally withdraw from the bloodstream and do not get replaced, the level of hemoglobin decreases (16). Reduction of hemoglobin concentration continues until the oxygen requirements of the tissues exceed the amount of oxygen delivered to the tissues. Naturally, this occurs between weeks 8 and 12, when the blood hemoglobin concentration is about 11 grams per deciliter. At this time, the production of erythropoietin is increased and blood transplantation continues. Even if you do not receive iron from your diet, the iron storage system of the **reticuloendothelial** system that results from the destruction of red blood cells is sufficient to re-synthesize hemoglobin to 20 weeks of age. This form of anemia is a physiological adaptation to ectopic life, which results in an increase in the amount of oxygen transferred to the tissues in excess of the amount needed. So, this form of anemia is not a hematologic problem and does not require treatment (17-19).

Bilirubin and its chemical properties

Bilirubin is a tetra-pyrrole pigment produced bythe destruction of the 'heme' part of aged red blood cells (91). It consists of four pyrrole Rings connected by three carbon bridges. Non-conjugated bilirubin in

physiological pH is almost insoluble in water, since COOH and NH-groups are involved in hydrogen bonding between strong molecules and, thus, not able to interact with water (20). These links are broken down by conjugation of the COOH groups with glucuronic acid. The reaction takes place in the liver cells, thereby potentially increasing the bilirubin molecule in water and changing its biological properties. Non-conjugated bilirubin is spreading almost beyond all biological membranes, such as blood-brain barrier, placenta, epithelium of the intestine and gallbladder, and only very small amounts are excreted intogallbladder. Therefore, liver conjugation allows bilirubin to be excreted from the body and prevent damage to the central nervous system. When non-conjugated bilirubin is exposed to light, polar optical isomers and lumirubin, which are the result of intracellular ringing, are formed. These compartments are eliminated without liver conjugation and thus are very effective in lowering the bilirubin concentration in hyperbilirubinemia and preventing its adverse effects (21).

Jaundice in infants

Neonatal Jaundice or Nebula Hyperbilirubinemia means yellowish skin color of newborns. All babies have some degree of bilirubin elevation (i.e., more than 2 mg/dL), but only half of the term babies are diagnoses with jaundice at levels up to 5 mg / dl (When bilirubin reaches 2 milligrams, jaundice becomes apparent in adults). A term newborn refers to an infant who has passes 37 weeks or more in her mother's womb. The frequency of jaundice in preterm infants reaches 80% of the total number of infants (22).

There are two main categories of jaundice in newborns, called indirect hyperbilirubinemia (increased levels of unconjugated bilirubin in the blood) and direct hyperbilirubinemia (increased conjugated bilirubin levels in blood); bilirubin removal, physiologic jaundice, carpal tunnel syndrome, Gilbert syndrome, jaundice of the breast, abnormality of the ABO and Rh blood groups, and glucose 6-phosphate dehydrogenase deficiency can be mentioned as the most common causes of indirect jaundice. Infection, cholestasis or liver cell damage may cause conjugated hyperbilirubinemia (direct), which has no neurological toxicity. Neonatal sepsis, neonatal metabolic diseases (such as gamma-lactose or tyrosinemia), deficiency of alpha-1 antitrypsin and L-syndrome are examples of diseases that cause direct hyperbilirubinemia. (23-25)

Indirect, non-conjugated Hyperbilirubinemia can be toxic and injurious to the brain and may lead to coronary heart disease, which is a lasting and severe injury to the neonatal nervous system. Various therapies such as phototherapy or blood transfusion might be needed to treat indirect hyperbilirubinemia (26).

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