CHARACTERISTICS AND TYPES OF HEMOLYTIC ANEMIA

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Annotation: *In this article, during the period of growth and development of Holstein calves, the daily feed of the Dutch state "Interchemie werken "De Adelaar" B.V." The procedure for using NEMOVIT WS - vitamin - amino acid nutritional supplement produced by the company, its norms, information about this nutritional supplement and the results that can be obtained when using it are covered on a large scale.*

Key words: *Animia, norm, growth, live weight, absolute growth, daily growth, relative growth, blood, hemoglobin.*

Medical Description - Anemia, by definition, involves a decrease in the level of red blood cells or hemoglobin. The term "hemolytic anemia" includes various types of anemia, where red blood cells are destroyed prematurely in the blood. The term "hemolysis" refers to the destruction of red blood cells (hemo = blood; lysis = destruction). Bone marrow has a certain reserve capacity. That is, the production of red blood cells can be increased to a certain level to compensate for their increase. Normally, red blood cells circulate in the blood vessels for about 120 days. At the end of their life, they are destroyed by the spleen and liver (see also Anemia page - overview). The rapid destruction of red blood cells is an important stimulus for the production of new red blood cells, mediated by the hormone erythropoietin (EPO), produced by the kidneys. In some cases, the bone marrow is able to produce an abnormally destroyed amount of red blood cells, so the hemoglobin level does not decrease. We are talking about compensated hemolysis without anemia. This is important because there are some factors that can cause the condition to be classified as factors that interfere with EPO production, such as pregnancy, kidney failure, folic acid deficiency, or acute infection.

Causes - Hemolytic anemia is usually classified according to whether the red blood cells themselves are abnormal (intracorpuscular) or caused by a factor outside the red blood cells (extracorpuscular). Hereditary and acquired hemolytic anemia are also distinguished. Hereditary and intracorpuscular causes

- Hemoglobinopathies (for example, sickle cell anemia, etc.).
- Enzymopathies (eg G6-PD deficiency)
- Membrane and cytoskeletal abnormalities (eg, congenital spherocytosis) Hereditary and extracorpuscular causes
- Familial hemolytic-uremic syndrome (atypical)
- Acquired and intracorpuscular cause
- Paroxysmal nocturnal hemoglobinuria

Acquired and extracorpuscular cause

- Mechanical disorder (microangiopathy)

- Toxic substances
- pharmaceutical products
- infections
- Immunological

Let's discuss a few examples, since it is impossible to describe all of them in the context of this document.

Immunological hemolytic anemia:

Autoimmune reactions. In this case, the body produces antibodies against its own red blood cells for various reasons: these are called autoantibodies. There are two types: those with warm autoantibodies and those with cold autoantibodies, whether the optimal temperature for antibody activity is 37 ° C or 4 ° C, this distinction is important because the treatment varies from form to form.

Warm autoantibodies: mainly affects adults and causes chronic and sometimes severe hemolytic anemia. They account for 80% of autoimmune hemolytic anemias. In half of the cases, they can be caused by certain drugs (alpha-methyldopa, L-dopa) or certain diseases (ovarian tumor, lymphoproliferative syndrome, etc.). These are called "secondary" autoimmune hemolytic anemias because they occur as a result of another disease.

Cold autoantibodies: Associated with acute episodes of cold-induced erythrocyte destruction. In 30% of cases, we deal with a secondary autoimmune reaction that can be explained by a viral infection or mycoplasma, an intermediate microorganism between viruses and bacteria.

Immunoallergic reactions. In immunoallergic (non-autoimmune) drug hemolysis, antibodies attack not red blood cells, but some drugs: penicillin, cephalothin, cephalosporins, rifampicin, phenacetin, quinine, etc.

Congenital hemolytic anemia: Red blood cells have three important components. Hemoglobin, a membrane-cytoskeleton complex, and an enzymatic "machinery" exist to make all of this work. A genetic abnormality in any of these three factors can cause hemolytic anemia. Hereditary abnormalities of the red blood cell membrane. The main one is congenital spherocytosis, so named because of the spherical shape that characterizes red blood cells and makes them particularly fragile. Relatively common: 1 in 5000 cases. Several genetic anomalies are involved, the classic form is autosomal dominant, but there are also recessive forms. This can cause certain complications: stones in the gall bladder, ulcers on the legs.

Enzymopathies. There are several forms of enzyme deficiency that can cause hemolytic anemia. They are usually hereditary. The most common is the lack of an enzyme called "glucose-6-phosphate dehydrogenase", which leads to early destruction of red blood cells and subsequent hemolytic anemia. The genetic defect is linked to the X chromosome, so only males can be affected. Women can carry a genetic defect and pass it on to their children. In people with this enzyme deficiency, hemolytic anemia often occurs after exposure to oxidants. People with G6PD deficiency may develop acute hemolysis under the influence of the following factors:

eating a variety of beans called fava beans (fava bean) or being exposed to pollen from that plant (this type of bean is used for livestock feed).

Such contact leads to acute hemolytic anemia called favism.

use of certain drugs: antimalarials, methyldopa (lowers blood pressure), sulfonamides (antibacterials), aspirin, non-steroidal anti-inflammatory drugs, quinidine, quinine, etc.

exposure to some chemicals, such as mothballs.

some infections.

This disease is most often diagnosed in people from the Mediterranean basin (especially the Greek islands), as well as in black people in Africa and the United States (where its prevalence is 10% to 14%). In some parts of the world, 20% or more of the population have it.

Hemoglobinopathies. This term is used to describe genetic disorders that affect the production of hemoglobin within red blood cells. Sickle cell anemia (sickle cell anemia) and thalassemia are two main categories of hemoglobinopathies. Sickle cell anemia (sickle cell anemia)4,5. This relatively serious disease is associated with the presence of an abnormal hemoglobin called hemoglobin C. This destroys the red blood cells and, in addition to causing their death, gives them a crescent or sickle (sickle) shape. ahead of time. See Sickle Cell Anemia.

Thalassemia. This serious disease, which is very common in some countries of the world, is associated with a genetic abnormality that affects the production of hemoglobin, which is the blood pigment in red blood cells that allows oxygen to be transported to organs. Affected red blood cells are fragile and break down quickly. The term "thalassemia" comes from the Greek word "thalassa" meaning "sea" because it was first observed in people from the Mediterranean basin. A genetic defect can affect two sites of hemoglobin synthesis: the alpha chain or the beta chain. Depending on the type of chain affected, there are two forms of thalassemia: alpha-thalassemia and beta-thalassemia. Other causes include:

Mechanical reasons. Red blood cells can be damaged during certain procedures involving mechanical devices:

prostheses (artificial heart valves, etc.);

extracorporeal blood purification (hemodialysis);

machine for filling blood with oxygen (used in cardio-pulmonary surgery) and others.

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