

ANEMIA AND HEMACHROMATOSIS

ANEMIA

This refers to shortage of RBCs or the content of Hb in them. This insufficient red cell mass can be the result of excessive destruction of RBCs (hemolysis i.e. hemolytic anemia), bleeding, blood disorders like thalassemia, or nutritional deficiencies e.g. iron, vitamin B12 (needed for the synthesis of Hb) deficiencies etc.

Hemolytic anemia occurs when red blood cells are being destroyed prematurely, due to a variety of reasons such as infections or certain medications — such as antibiotics or antiseizure drugs etc in autoimmune hemolytic anemia, the immune system mistakes RBCs for foreign invaders and begins destroying them. Blood disorders such as thalassemias, hemoglobinopathies can also result in rapid destruction of RBCs.

Bleeding or blood loss can also cause anemia and maybe because of excessive bleeding due to injury, surgery, cancers or a problem with the blood's clotting ability.

Inadequate production of RBCs is also another major cause of anemia and this could possibly be due to nutritional deficiencies e.g. iron deficiency anemia, the most common cause of anemia in piglets. Or it maybe due to problem with the bone marrow due to a viral infection, or exposure to certain toxic chemicals, radiation, or medications (such as antibiotics, antiseizure drugs, or cancer treatments), or as a result of kidney failure (produces erythropoietin).

SIGNS

The first symptoms might be mild skin paleness and decreased pinkness of the mucous membranes. Irritability, fatigue, weakness and a rapid heartbeat. If the anemia is caused by excessive destruction of RBCs, symptoms also may include jaundice, a yellow discoloration of the mucous membranes. Decreased appetite, blood in the urine or feces, an enlarged spleen, abdominal distension and dark tea-colored urine may also be seen.

HEMACHROMATOSIS

This is a disorder of iron metabolism as a result of excess iron absorption, saturation of iron binding proteins and deposition of hemosiderin (amorphous iron deposits in cells, composed of ferritin, denatured ferritin, and other materials with its molecular structure poorly defined in tissues). Primarily affected are liver, pancreas, skin and can lead to cirrhosis of the liver and diabetes (when the pancreas is affected) and bronze pigmentation of the organs and skin.

The bronze pigmentation and resulting diabetes warrants the designation of the disease as bronze diabetes.

The condition is primarily genetic due to inheritance of an autosomal recessive allele. HFE gene (a histocompatibility complex gene) regulates iron transfer into cells via its formation of complex with transferrin hence a mutation in this gene results in abnormal iron intake and storage. Secondary hemachromatosis which is not genetic can result from excess oral intake of iron or in patients receiving blood transfusion.