CHARACTERISTICS, DISEASES AND DIAGNOSIS OF IRON METABOLISM-HEMOSIDERIN

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Annotation: This article provides information on the properties of iron metabolism-hemosiderin, diseases and diagnostics

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Hemosiderin is a pigment in the form of lumps or granules stored in the tissues of iron animals. These granules are poorly assimilated in the body, they are stored inside the cells and usually appear after heavy bleeding.

Hemosiderin corpuscles have poorly defined molecular properties despite their iron nature. However, they are known to consist of ferritin, denatured ferritin, and other materials. In addition, hemosiderin granules are always opposite or opposite to blood flow. Hemosiderin is often found in macrophages called “siderophages”. These are macrophages that are responsible for phagocytosis in red blood cells (erythrocytes) and because of this phagocytosis iron is released in them and it is stored in an organelle called a siderosome.

Siderophages are cells produced by the bone marrow that are responsible for storing iron to deliver to red blood cells during the formation of red blood cells (erythropoiesis). The appearance of siderophages indicates bleeding due to certain pathological agents or some mechanical stresses. Siderophages usually appear 48 hours after bleeding and can persist for 2-8 weeks after bleeding.Hemosiderin is detected by blood smears, tissue samples, or substances taken from different regions of the body. These blood samples are treated with staining methods, where siderophages are easy to identify due to their size and intense blue color. Hemosiderin represents a set of structures that act as intracellular iron stores, are water-insoluble, and are stored in phagocytes of the spleen, liver, and bone marrow reticulum endothelial system. Each hemosiderin granule can contain up to 4,500 iron atoms.

Iron stored in hemosiderin granules is considered to be iron phosphate. This compound is a major component of cellular iron stores in the form of ferritin.

However, ferrite-shaped iron deposits are much smaller and more assimilated by cells than hemosiderin granules. Cells containing ferritin also observed the presence of hemosiderin granules.

50% of the constitution of hemosiderin deposits consists only of iron atoms.

Scientists who observed hemosiderin grains under an electron microscope found that they were ferritin, denatured ferritin, a complex of proteins, carbohydrates, lipids, and other materials.

The size of hemosiderin granules can range from 1 nanometer to 20 nanometers, which are large crystals or granules. They think that it can be assimilated by the cell only through lipid peroxidation under the influence of iron.
Hemosiderin has been proposed to demonstrate a “protective” biological mechanism because it reduces the presence of iron, which enhances reactions caused by free radicals within cells.

The full functioning of iron regulatory mechanisms in the body of animals is very important for health because iron deficiency causes anemia; iron overload in the system promotes the accumulation of hemosiderin in the tissues.

Such accumulation of hemosiderin can cause tissue damage and lead to a condition called “hemosiderosis”. The disease is characterized by cirrhosis of the liver, possibly accompanied by liver carcinomas.

The defective hemochromatous locus may indicate defects in the HLA-A mucosal regulation system in the short arm of chromosome 6, as if there was persistent iron deficiency even when the mineral was ingested in abundance.

This disease can take two forms, primary or secondary hemochromatosis. Primary hemochromatosis is an autosomal recessive disease. In this case, people tend to keep iron uncontrolled in the form of hemosiderin in the tissues.

However, primary hemochromatosis can be managed by blood transfusion and blood draw. If the disease is diagnosed early, until excessive accumulation of hemosiderins in human tissues occurs.

Secondary hemochromatosis occurs when the iron regulatory system is overloaded with iron due to the death and extinction of erythrocytes, liver disease, or chronic iron overload.

Hemosiderins have been diagnosed from different perspectives. For pathologists, they are iron-containing fragments, and for biochemists, they are heterogeneous compounds of iron, carbohydrates, proteins, and lipids.

For electron microscopists, hemosiderin bundles are electron-dense compounds located inside siderosomes (bodies that carry pigments).

However, despite the different positions on hemosiderin granules, they all agree that they are insoluble granules rich in iron and that their excess is harmful to the health of the organism.

Hemosiderin granules form particularly large fragments in cells and can be easily stained in tissues to see them clearly under a light microscope.

Hemosiderin granules were stained with a Prussian blue reaction using a method called Perl stain. Using this technique, the differences between isolated hemosiderin iron cores with different conditions are described, e.g.

- The hemosiderin nuclei of patients with secondary hemochromatosis have a goetite-like crystalline structure with the chemical formula a-FeOOH.

- In patients with primary hemochromatosis (genetic origin), the iron nucleus of hemosiderin granules is amorphous, consisting of iron III oxide.

In normal human spleen cells, which store iron in some hemosiderin granules, the nuclei appear to be crystalline ferhydrite, very similar to the nuclei of ferritin molecules.

Using an electron microscope, a more detailed diagnosis can be made to differentiate patients with primary hemochromatosis and secondary hemochromatosis.

Typically, hemosiderin particles range from 5.3 to 5.8 nanometers in people with primary hemochromatosis; however, in patients with secondary hemochromatosis, they measure measurements ranging from 4.33 to 5 nanometers in diameter.

These data are important to determine the type of disease in patients. In addition, genetic analysis confirms the genetic composition of the body's cells in the tissues of this disease.

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