Національний університет біоресурсів і природокористування України

Факультет ветеринарної медицини

НДІ Здоров'я тварин

«ЄДИНЕ ЗДОРОВ'Я – 2022»

Матеріали Міжнародної наукової конференції

22-24 вересня 2022 р.
НУБіП України, м. Київ

UDC:619:612.3:636

CLINICAL AND BIOCHEMICAL INDICATORS FOR HYPOCHOLESTEROLEMIA AND HYPERCHOLESTEROLEMIA

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Cholesterol is a waxy, fat-like substance that's found in all the cells in a body. Exogenous and endogenous cholesterol are distinguished by their origin in the body. Exogenous enters the body with food of animal origin, and endogenous is synthesized mainly in the liver (up to 80%), enterocytes, the outer layer of the adrenal glands, muscles, brain and other tissues. Cholesterol is synthesized from hydroxymethylglutaryl-CoA in many tissues, but mostly in the liver and intestines. In the form of an ester with an unsaturated fatty acid, it forms cholesterol esters.

Cholesterol is part of cytoplasmic membranes and membranes of organelles, and is also a precursor of steroid hormones and vitamin D. Bile acids and steroid hormones of five classes are formed from cholesterol: progestogens, glucocorticoids, mineralocorticoids, androgens, estrogens. Cholesterol enters the blood in the form of chylomicrons (from the gastrointestinal tract through the lymphatic system) and lipoproteins VLD and LD (from the liver and other organs). Disruption of cholesterol metabolism in monogastric animals is caused by an imbalance between its intake with feed, synthesis in the body and removal from the body. These disorders are manifested in a change in the total cholesterol content both in the whole body and in individual organs and tissues. Hypercholesterolemia is divided into primary (alimentary) and secondary, caused by various diseases.

The cholesterol content in the blood depends on the physiological state of the animals. The upper limit of the norm in young animals is 2-3 times lower than in adults. Hypercholesterolemia occurs as a result of several reasons, including: increased mobilization of lipids from depots and their catabolism; excessive intake of exogenous cholesterol; violation of bile formation and bile secretion; violation of cholesterol utilization.

It is noted for stress, the initial period of starvation, diabetes, myxedema, mechanical jaundice, nephrotic syndrome, chronic renal failure and atherosclerosis.

Hypocholesterolemia occurs as a result of impaired synthesis of endogenous cholesterol in the liver, nutritional deficiency, and increased cholesterol utilization. Hypocholesterolemia accompanies hyperthyroidism, various forms of anemia, cachexia, liver diseases characterized by necrosis of hepatocytes – toxic hepatitis, malignant tumors and invasive liver diseases. In the case of an acute course of hepatitis, the level of total cholesterol is rise (hypocholesterolemia).

Cholesterol metabolism disorders play a very crucial role in gallstone disease. This is a disease associated with the appearance of hard concretions - gallstones in the biliary tract, which cause a violation of the outflow of bile from the biliary tract or the appearance of an inflammatory process in them. Usually, the main mass of gallstones consists of cholesterol and bilirubin salts. If more than 70% of its weight is cholesterol in the composition of stones, then these stones are called cholesterol stones (diagnosed in 2/3 of cases). Normally, excess cholesterol is excreted from the body, mainly with bile. Cholesterol is difficult to dissolve in water, so it is in bile as part of micelles that ensure its dissolution. Micelles also include bile acids and phospholipids (phosphatidylcholine or lecithin), which ensure the solubility of cholesterol in the aqueous phase of bile. Hepatocytes secrete cholesterol in micellar form. Bile from the liver enters the gallbladder, where it is concentrated due to the reabsorption of part of the water through the bladder wall. At the same time, there is an increase in the relative concentration of cholesterol compared to the concentration of bile acids. If this process leads to a disruption of the structure of the micelles, the conditions are created for the transition of cholesterol from the micellar stable form to the liquid crystalline form, which is unstable in water. During the progression of this process, the transition of cholesterol into a solid crystalline form occurs, which leads to the formation of cholesterol stones. In case of violation of bile formation directly in the liver, cholesterol crystals may appear in the bile even before it enters the gallbladder. This is due to a significant excess of cholesterol entering the bile, or to a decrease in the amount of bile acid synthesis. Biochemical markers of gallstone disease in the blood are an increase in the level of triacylglycerols, pre-β - and β-lipoproteins, and cholesterol. The biochemical properties of bile change significantly: the concentration of bile acids decreases with a simultaneous imbalance of their types, the content of proteins, bilirubin, and lipids increases, and the content of glycosaminoglycans, which are inhibitors of stone formation, decreases.