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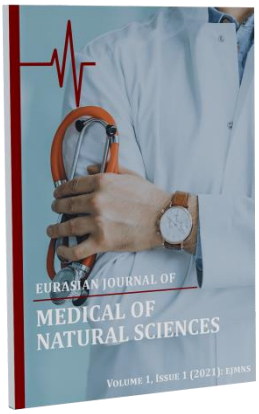

THE COURSE OF CARDIOMYOPATHY IN PUREBRED CALLAMUSES

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ABSTRACT

The heart belongs to the order of the organ, which participates in the movement of blood circulation and lymph in the body, manifests itself in an empty muscle organ. The heart of mammals: the base and upper part, the left and right surface, the front and the middle edge are separated into parts. The walls of the heart are formed from the endocardium, myocardium, and epicardium. The endocardium is the inner shell of the heart, which is made up of connective tissue and is covered with endothelium. The myocardium is the middle shell of the heart, formed from muscle fibers of various orders. The anterior division of the heart and ventricles are separated by myocardial fibrosis rings in a non-interfering manner. The epicardium was manifested by the outer serous shell of the heart.

The term" cardiomyopathy " was first coined by W. Brigden used it as early as 1957, and it is primarily used to define conditions affecting heart muscle without inflammation. Cardiomyopathy can be classified into idiopathic (i.e. with an unknown etymology; it is sometimes referred to as primary) or dichotomous. Idiopathic cardiomyopathy can be classified into lower classes based on pathological anatomy and physiology – dilatasialy, hypertrophic and intergrad.

Dilated cardiomyopathy (DKMP) is a disease characterized by primary myocardial damage, expressive dilation of cavities, as well as impaired systolic function of the ventricles. The term DKMP is used in cases of heart lesions where only a large amount of dilation of cavities is not considered a consequence of coronary circulation disorders, congenital anomalies of development, valve defects of the heart, systemic and pulmonary arterial hypertension and pericardium disease.

It has been scientifically proven that pedigree and broodless callamush are susceptible to DKMP. DKMP is found in most cases in pedigree callamushes, they are sick 2-3 times more often than in non-pedigrees. The diagnosis of this pathology is often made in middle (6 months old) and elderly (older than 9 months old) callamushes.

Dilated cardiomyopathy is both a primary and acquired heart disease of the heart in many purebred callamushes. Genetic etiology has been isolated for primary DKMP. But it is known that some other causes are able to call the development of DKMP, in this case, the development of the disease is introduced into secondary etiological factors. The most likely causes that lead to the occurrence of the disease are: food, inflammation, infection, infiltrative, ischemic and medicinal or toxic diseases of the myocardium; tachycardia-induced cardiomyopathies; metabolic disorders; genetic-biochemical defect of the animal organism; previously identified viral (e.g., entrovirus) infection; immunological disorders; chemical toxins, amino acid deficiency are considered.

Under the influence of exogenous factors, the number of fully functional cardiomyocytes decreases, which leads to an expansion of the heart chambers as well as a violation of the myocardial contraction function. The heart cavities expand, which leads to the development of systolic and diastolic dysfunction in both ventricles, which creates conditions for the development of chronic heart failure. In the early stages of the disease, Frank-Starling's law works, which states that the degree of diastolic stretch is proportional to the force of contraction of myocardial fibers. The blood drive of the heart is also maintained at the expense of an increase in the frequency of heart contractions and a decrease in peripheral resistance during physical loading. Increasingly, compensatory mechanisms are disrupted, the rigidity of the heart increases – by contracting, the heart muscle will not be able to contract repeatedly, systolic function deteriorates, and the Frank-Starling law will stop working.

The minute and tattoo size of the heart decreases, the final diastolic diameter of the left ventricle becomes smaller, and further expansion of the heart cavities occurs. Due to ventricular dilation, relative insufficiency of mitral and three-stvor valves as well as expansion of Valve rings occurs. In response, compensatory myocardial hypertrophy occurs, which also leads to a decrease in Wall loading as well as a decrease in Gap dilation, as a result of which there is myocyte hypertrophy as well as an increase in the volume of connective tissue. A decrease in the volume of blood flow to the heart as well as an increase in intra-ventricular diastolic pressure can lead to a decrease in coronary perfusion, the consequence of which will be subendocardial ischemia. Tissue oxygenation is reduced by stopping a small circle of blood circulation in the veins.

DKMP is a disease that can be prolonged subclinical (unmarked), the clinical signs of which are manifested only in the terminal stage of heart failure. Clinical Anamnesis is often very short, covering a period of several days to a week, while the common, most common clinical symptoms are animal apathy (lethargy), weight loss, cough, mixed various shortness of breath, decreased susceptibility to physical loads, anorexia and abdominal pus (assitis).

The volume of blood flow to the heart as well as a decrease in ventricular contraction leads to a sluggish or alternating pulse, compensatory tachycardia and poor Peripheral Perfusion, as a result of which mucosal anemia and a decrease in local temperature in the area of the limbs develop.

The origin of the cough is due to the excitation of baroreceptors to the location on the wall of the head bronchi, which are compressed by an enlarged heart in size. Enlarged and enlarged ventricles slowly receive nerve impulses passing through the anterior section of the heart, causing a presystolic rhythm of jumping out in auscultation. In addition, the runctum

optimum of the mitral and tricuspidal valves (p. o.) can hear endocardial noise caused by regurgitation of blood from the ventricle to the anterior section of the heart, but these noises are difficult to detect due to a violation of the general rhythm of the work of the heart.

Hypervolemia as well as a decrease in heart muscle contraction lead to pulmonary edema, as a result of which tachypnoe and mixed-type shortness of breath are formed.

Kovaleva S. P. and hammual. (2013, 2019), Tsherbakova G. G. and hammual., (2020), Stekolnikova A. A. and hammual. (2011, 2017, 2018) et al. according to the data given, the development of right ventricular insufficiency often leads to edema of the yoke veins, positive venous pulse, hepatomegaly and assitis.

In a macroscopic study of the patient's heart containing DKMP, it is noted that the weight of the member has significantly increased. In addition, the expressive dilation of all spaces, the whiteness and emptying of the myocardium are considered signs characteristic of it. A significant increase in heart mass and heart index occurs mainly at the expense of the ventricles, especially the left ventricle. At the same time, the colon of the wall of the ventricles and the wall between the ventricles changes relatively little, which is due to the fact that the expression of hypertrophy is dominated by the dilation of the cavities. Many authors note that dilation of all cavities in the heart is found in an average of 85% of callamushes with DKMP.

Notable is the change in cardiac configuration close to the sharsimon. Thus, in macromorphometric analysis, it is observed that its greatest circumference, width and width at the base, or a significant enlargement of the anterior-posterior dimension, in which the length, that is, the distance from the base to the summit, practically does not change.

When an autopsy is performed, usually no major changes are detected in the endocardium. Its imperceptible coughing is usually recorded only in the area of the left ventricle. Often, Mural thrombosis is observed, which in most cases serves as a source of thromboembolism within the framework of large and small blood circulation. The reasons for the formation of thrombosis in front of the wall are a slowdown in blood flow caused by disturbances in their relaxation in the cavities of the heart, as well as a hypercoagular orientation of hemostasis changes. Most often, thrombi settle in the ventricles, mainly in the left ventricle, which causes endocardial coughing.

When light microscopy is performed, two types of histological changes are observed in the myocardium. They are named as an attenuated wavy species and an oily infiltration-degenerative species. Myocardial lesions associated with the attenuated wavelet type of DKMP are made up of myocardial cells less than 6 μm in diameter (the normative diameter of myofibrils ranges from 10 to 20 μm (Van Fleet, J., Ferrans, V., 1995)), has a wavy exterior, and the left ventricle accounts for less than half of the myocardial sample Colon in the upper and lower wall sections. Myocytes are clean, usually separated from cell infiltrates by a free sphere. Diffuse infiltration of subendocardial fibrosis is also observed in many cases.

Myocardial damage associated with the fatty infiltrative-degenerative type of DKM includes myocytolysis, myofibrillar degeneration, myocyte vacuolization with extensive myofibrillar displacement fibrosis and oilinfiltration. Fibrosis-precipitation of myocardial tissue is seen as a consequence of loss of myocytes due to various causes, such as myocarditis or other harmful triggers. A number of anti-tumor agents, such as doxorubicin and cyclophosphamide, can call for extensive vacuolization of myocytes.

A weak wave fiber type of DKMP, a. Tidholm and L. Jonsson (2005) states that it is the most common form.

The contractile myocardium has not changed in area and volume, and its hypertrophied cells are surrounded by a large number of atrophied cardiomyocytes, which are located everywhere and are detected in different sections of the heart muscle.

Interstitial fibrosis, as well as an increase in the connective tissue component at the expense of replacement sclerosis, is a specific aspect. The cells and fibers of the connective tissue are located intramiocardially on the walls of all four chambers of the heart, mainly in the inner third of the myocardium, without attachment to the vessels. The specific rate of sclerosis muscle fibers, in which the connective tissue is expressed to varying degrees, spread to the Massif, represents the stage of cardiomyocyte destruction. Dkpm in existing callamushes, the prognosis depends on what stage the animal is in the development of the disease. When the decompensator is in the dkmp stage, M. Martin et al. (2001) found that the prognosis is negative. The prognosis often depends on the breed of the callamush. In general, the average duration of life after diagnosis was calculated to be about six months. In the opposite case, death occurs as a result of ongoing uncontrolled stagnant heart failure.

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