THE MORPHOPATHOLOGICAL PREVALENCE OF INFLAMMATORY CARDIOMYOPATHIES IN DOGS

ADRIAN OLARIU-JURCA
Banat's University of Agricultural Sciences and Veterinary Medicine „King Michael I of Romania" from Timisoara, Romania
olariujurca_adrian@yahoo.com

Abstract: *The research was conducted during the period October 2013-May 2015 and it consisted of the necropsic exam of 30 dog bodies of different ages, sexes and breeds. It was completed with a microscopic exam of the histopathological preparations which were obtained from heart samples coming from 19 cases which presented lesions. Eight cases were diagnosed with inflammatory cardiomyopathies (42.09%). The diagnosed cardiac lesions, of the cases taken into study were multiple and complex, they developed in the same time or successively in several structures (pericardium, myocardium and endocardium as well as in blood vessels). They were exteriorized through pericarditis (5.26%), myocarditis (21.5%) and endocarditis (10.52%) as well as parasitic pulmonary arteritis (5.26%). Fibrinous pericarditis, chronic evolution, signaled in one of the cases was histopathologically exteriorized through the presence of fibrin networks with leukocytes and erythrocytes, attached to the swollen and infiltrated with inflammatory cells epicardium.*

The myocarditis which was morphopathologically identified, was represented by the parenchymatous myocarditis in one case, lymphohistiocitary myocarditis in two cases and fibrous interstitial myocarditis in one case.

The endocarditis we diagnosed morphopathologically was represented by the verrucous or thrombotic endocarditis (5.26%) as well as by ulcero-polypous endocarditis (5.26%).

In one case with cardiopulmonary dirofilariosis, we anatomopathologically diagnosed wormy pulmonary arteritis, expressed through lesions of several degrees, intensities and extensions of the arterial wall. They were translated through erosions of the intima as consequence of the contact with dirofilaria larvae.

Key words: *morphopathological prevalence, inflammatory cardiopathies, dogs*

The heart, respectively the myocardium, is the biggest energy consumer in the animal organism during their entire lives. In this purpose, the cardiomyocytes, despite the fact that in essence they are only muscular fibers, possess an ultrastructure which is orientated towards the oxidative processes. It is considered that the number of the mitochondrias is around 250 billion/ myocardium gram. They are big oxygen consumers, 5 times bigger than the cells in the liver (7).

Under the influence of numerous aggressors, cathecolamines accumulate in large quantities and cause a short intensification of the metabolic activity. This generates and oxygenation deficit, morphopathologically translated through fuchsinephile degeneration, myocytolisis followed by the activation of the active mezenchyma, hyperplasia and differentiation in the directions of cellular and humoural defense with the apparition of a reactive complex with different influences on the vital functions (2, 4, 9, 10).

The lesions of the myocardium start in the shape of relatively simple modifications which amplify and complicate in time, leading to more or less serious heart failure (1, 2, 4, 7).

Heart diseases generally have circulatory disorders as pathomorphic substrate (thrombosis, ischemic necrosis), atrophies, specific and unspecific inflammations, processes

that do not differ significantly from the ones located in other places. The only differences regard etiological conditions and specific clinical manifestations (3, 5, 6, and 10).

MATERIALS AND METHODS

The research of the present study was conducted in the period October 2013-May 2015, through an anatomopathological examination of hearts taken from 30 dog bodies of different ages, breeds and sexes, 19 cases presented cardiac lesions. The bodies came from private owners, kennels and animal shelters and they were necropsied and examined at the Forensics discipline in the Faculty of Veterinary Medicine.

The bodies were put in dorsal position and then they were necropsied. After the external and internal examination of the bodies we performed the evisceration according to the specific technique (5, 6). The evisceration of the heart was made by sectioning the sterno-pericardial ligament and the big vessels. This was followed by the macroscopical examination of the heart through inspection, palpation and sectioning in order to identify the physic-structural modifications of the heart 9 shape, size, colour, aspect, and shine, thickness of the pericardium, myocardium, and cavity ratio.

After a detailed examination of the heart, 19 samples were taken from the bodies which presented cardiac lesions, from the: pericardium, myocardium, endocardium and pulmonary artery for the histopathological exam. The samples were processed using the paraffin technique; they were cut at 6 μm and stained using the HEA method in order to identify microscopic cardiac lesions. The histopathological preparations thus obtained were examined using a research microscope MC 5 A with ocularies of 10 and higher and the photographing was done using an Olympus CX41 microscope (acquired through POS CCE, DICES-MVT 2669-145). After the examination and interpretation of the histopathological preparations, microphotographs were taken to illustrate the most characteristic histological modifications.

RESULTS AND DISCUSSIONS

The research regarding cardiomyopathies in dogs was systemized intro four groups: pericarditis, , myocarditis, endocarditis and arteritis (Table 1, Fig.1.).

The inflammatory cardiomyopathies were diagnosed in 4 cases out of which one was parenchymatous.

Myocarditis, two cases were lymphohistiocitary myocarditis and one was fibrous interstitial myocarditis.and with friable consistency. The serous pericardial surfaces (visceral and parietal are thickened, opaque, dull, harsh due to fibrin deposits (hairy heart). Fibrinous pericarditis with chronic evolution was diagnosed in the case which presented interstitial fibrous myocarditis.

Macroscopically, the pericardial sack is distended and slightly adherent to the epicardium due to the fibrinous exudate, of grey-yellowish colour, with an aspect similar to greaves Myocarditis, two cases were lymphohistiocitary myocarditis and one was fibrous interstitial myocarditis.and with friable consistency. The serous pericardial surfaces (visceral and parietal are thickened, opaque, dull, harsh due to fibrin deposits (hairy heart). Microscopically, using the x10, x20, x40 objectives we noticed: the myocardium in transversal section, subepicardial conjunctive tissue, swollen epicardium, infiltrated with leukocytes and erythrocytes, fibrin network attached to the epicardium,

with leukocytes and erythrocytes in its holes, with fibrinoidosis and coagulation necrosis of the valvular endocardium (Fig. 2.).

The parenchymatous myocarditis or the acute toxic myocarditis was identified in one case, a stray, 4 year old, male dog from a kennel. Macroscopically, the heart was enlarged, of grey-yellowish color, even both at inspection and on section, with the aspect of a boiled organ. This type of inflammation is hard to differentiate macroscopically from the granular myocardosis, the only exam capable of differentiating them being the histopathological exam. Microscopically, we noticed granular degeneration in the myocardiocytes, hyalinosis in the myocardial fibers, sero-leukocytic interstitial exudate and rare fibrilar hyperplasia of the local mezenchyma in the perifibrilar and peri-fascial spaces (Fig. 3.). This inflammation is the consequence of various intoxications and also of canine parvovirus and herpesvirus. Lymphohistiocitary myocarditis, found in two cases, was exteriorized macroscopically through the enlargement of the heart, even grey-yellowish color and on section the myocardium had a fatty aspect and high consistency. Microscopically, we could notice interstitial, lymphohistiocitary hyperplasia, located perifibrilar and perifascial, fallowed by atrophies and dystrophies and necrosis of the myocardial fibers (Fig. 4.). It is signaled in parvovirus encephalo-myocarditis in puppies. It is more frequent in young puppies from unvaccinated others and in dogs up to the age of 8 weeks is caused by a parvovirus.

Fibrous interstitial myocarditis, diagnosed in one case, is a predominantly productive inflammation that appers either as a consequence of necrotic, parenchymatous or lymphohistiocitary myocarditis or as an irrigation disorder of the myocardium. Macroscopially, on inspection and on section, diffuse white areas of fibrosis, of high consistency, can be noticed on the red-brownish background of the myocardium. Microscopically, there are exudates, infiltrations of inflammatory cells, mostly eosinophils and peri-fibrilar and peri-fascial fibro-conjunctive hyperplasia which lead to atrophies, dystrophies, and necrosis of the myocardial fibers (Fig. 5.).

Verrucous endocarditis (thrombotic) diagnosed in one case, is macroscopically exteriorized through the apparition on the tricuspid valvular endocardium of deposits with an irregular surface, isolated or confluated, of various dimensions (Fig. 6.).

Microscopically, we noticed endothelial desquamation, agglutination of thrombocites and deposition of fibrin in the shape of polyps (Fig.7.). Ulcero-polypous endocarditis was identified in one case, with right cardiac dilation. Macroscopically, one can notice voluminous polyps on the valve endocardium, with an irregular surface, altenated with ulcerative areas (Fig. 8.). Microscopically, the process starts with an ulceration, with abundant fibrin and cellular deposits on its surface (mixed thrombi). One can notice: sero-fibrinous and leucocitary infiltration of the endocardium, polyps made of fibrin, leukocytes and red blood cells, fibrinoidosis and coagulation necrosis ; reduced unspecific mesenchymal reaction which justifies the high friability of the polyps (Fig. 9.).

Verminous pulmonary arteritis was identified in one case which presented cardiac dirofilariosis (Fig. 10.). Microscopically, there are lesions of different degrees of intensity or extension in the arterial wall. They are translated through erosions of the intima as consequence of the contact with dirofilaria larvae (L5). Vascular lesions are represented by endothelial denudation, larvae thromboembolisms, endarteritis with

leukocitary infiltrate, mostly eosinophils, intimal hyperplasia with intraluminal focal appearance, giving it its rough aspect and in several areas, there are also granulomatous reactions in the arterial wall (Fig. 11.). The vascular lesions are the effect of mechanic trauma caused by parasites, but also of the intervention of several cytokins, especially the growth factor derived from the plates. This hypothesis is also mentioned in the consulted specialty literature. The intimal roughness favors obliterant thrombo-embolisms which cause pulmonary infarctions. The acute inflammatory reaction lasts as long as the larvae are present, and are accompanied by proliferation of the conjunctive tissue which will organize the superficial thrombus.

Table 1

Frequency of cardiomyopathies

No.	Necropsied dogs	Total of cases with cardiomyopathies	Inflammatory cardiomyopathies								%
			Pericarditis		Myocarditis		Endocarditis		Arteritis		
			%		%		%		%		
1	30	19	5,26	1	21,05	4	10,52	2	5,26	1	42,09

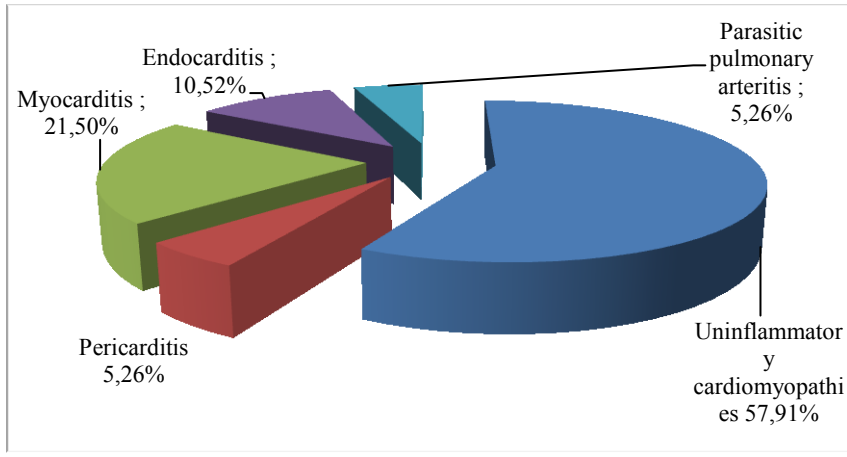

Fig. 1. Graphic representation of the prevalence of inflammatory cardiomyopathies.

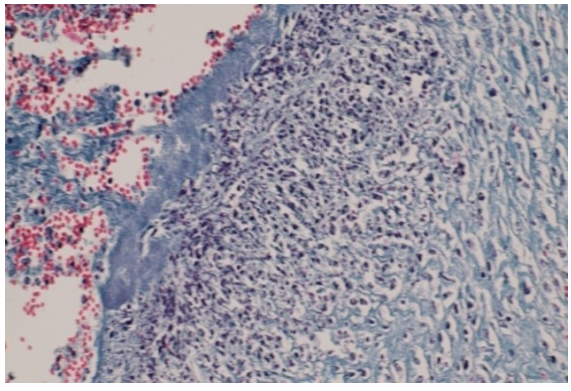

Fig. 2. Fibrinous pericarditis: swollen epicardium, infiltrated with leukocytes and erythrocytes, fibrin network attached to the epicardium, with leukocytes and erythrocytes. Col. HE x 10.

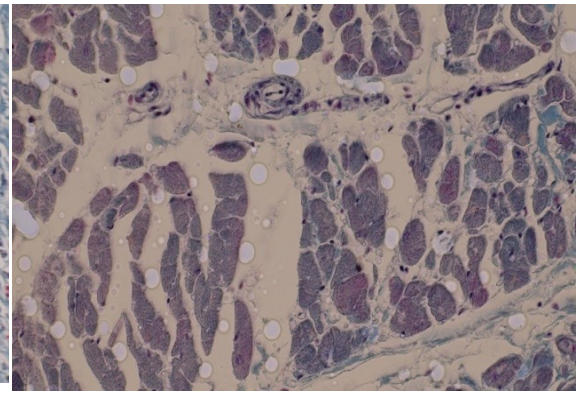

Fig. 3. Parenchymatous myocarditis: granular degeneration in the myocardiocytes, hyperplasia of the local mezenchyma in the perifibrilar and peri-fascial spaces. Col. HEA x40.

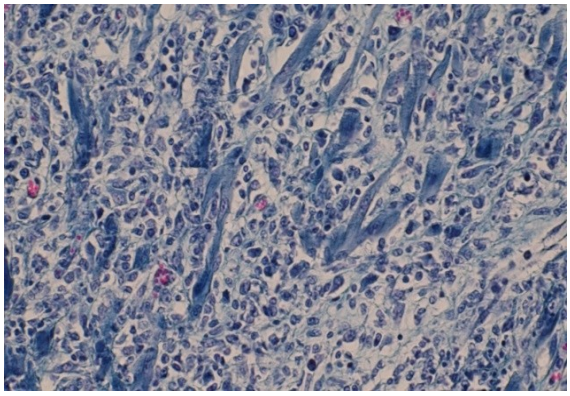

Fig. 4. Lymphohistiocitary myocarditis: lymphohistiocitary hyperplasia, located perifibrilar and perifascial fallowed by atrophies, dystrophies and necrosis. Col. HEA x40.

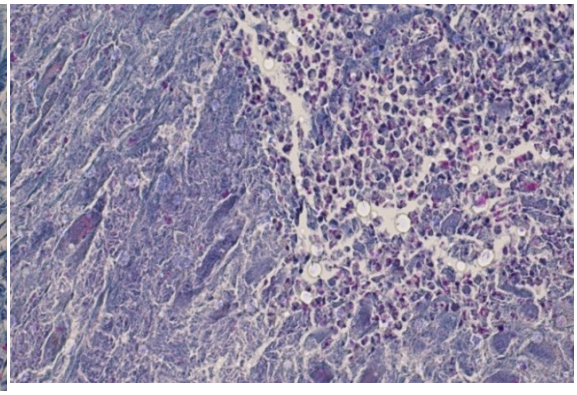

Fig. 5. Fibrous interstitial myocarditis and eosinophilic: interstitial fibro-conjunctive and eosinophilic hyperplasia which lead to atrophies, dystrophies, and necrosis. Col. HEA x20.

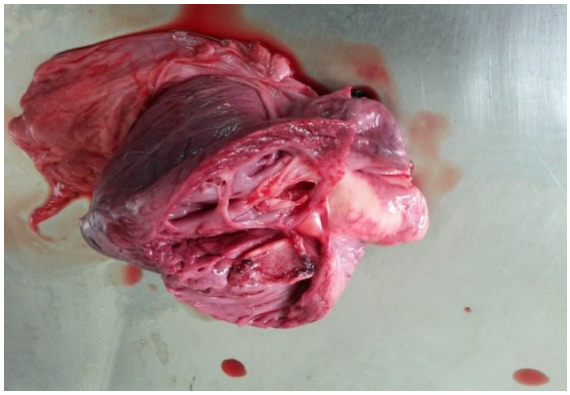

Fig. 6. Verrucous endocarditis (thrombotic): deposits with an irregular surface, isolated or confluated on the tricuspid valvular endocardium.

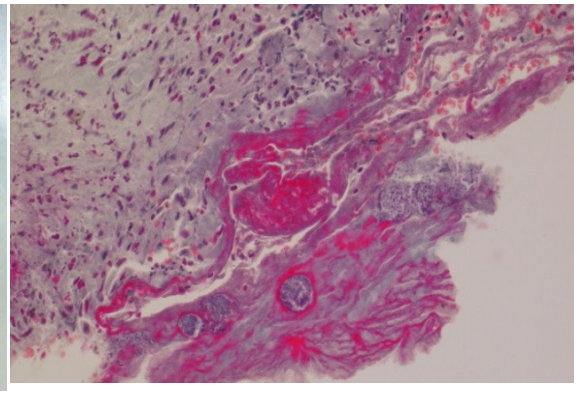

Fig. 7. Verrucous endocarditis (thrombotic): thrombus attached to the swollen and edematous endocardus. Col. HEA x40.

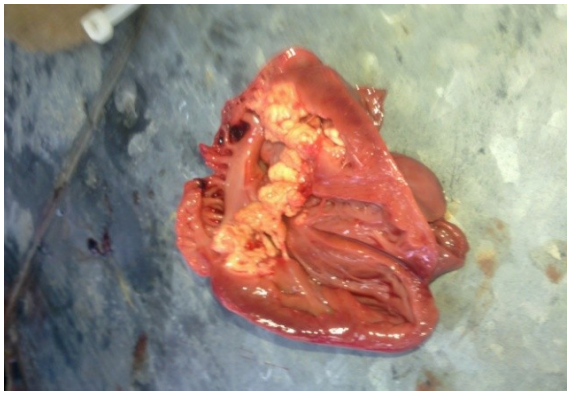

Fig. 8. Ulcero-polypous endocarditis and myocardial steatosis.

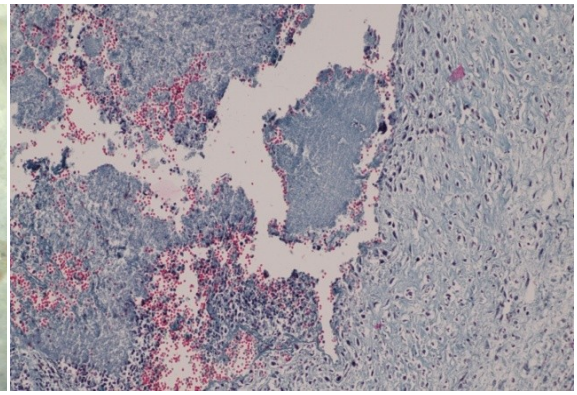

Fig. 9. Ulcero-polypous endocarditis: vegetation attached to the edematous endocardium infiltrated with inflammatory cell elemets. Col. HEA x 20.

Fig. 10. Dirofilaria in cardiac cavities and pulmonary artery.

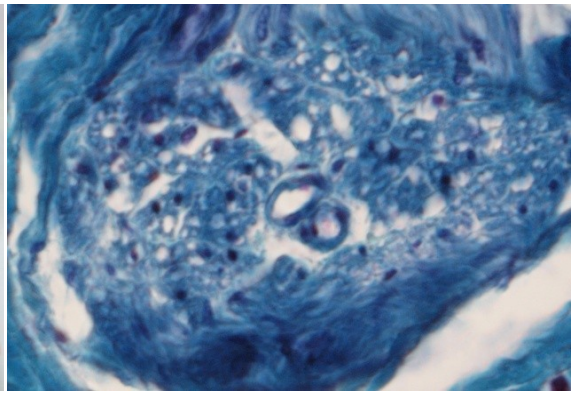

Fig. 11. Vermin pulmonary arteritis (dirofilaria): endothelial denudation and larval thromboembolisms. Col. HEA x 100.

CONCLUSIONS

Out of 19 cases presented, which have shown lesions of the heart, 8 cases were diagnosed with inflammatory cardiopathies.

The hearts belonging to the examined cases have shown inflammatory lesions (42, 09%), proving a big implication in the cardiac pathology of this species.

The diagnosed cardiac lesions, of the cases taken into study were multiple and complex, they developed at the same time or successively in several structures (pericardium, myocardium, endocardium and blood vessels) exteriorized through pericarditis (5.26%), myocarditis (21.5%), endocarditis (10.52%) and pulmonary arteritis (5.26%). The inflammatory myocardiopathies are on the first place, and they are represented by parenchymatous ar acute toxic myocarditis (5.26%), lymphohistiocitary myocarditis (10.52%) and fibrous myocarditis (5.26%), lymphohistiocitary myocarditis (10.52%) and fibrous myocarditis (5.26%) followed by thrombotic or verrucous endocarditis (5.26%), ulcero-polypous endocarditis (5.26%) and pulmonary verminous arteritis (5.26%).

The vascular lesions are represented by the verminous thrombo-arteritis of the pulmonary artery, caused by dirofilaria.

REFERENCES

1. BRĂSLAȘU, C.M., (2011)- Bolile cordului în medicina internă a animalelor, coordonator FALCĂ,C, Editura Eurostampa, Timișoara, cap XXIV,582-643.
2. CALIARI, M.V., MACHADO, R. DO., LANA, M. DE., CAJÂ, R.A.F., CARNEIRO, C.M., BAHIA, M.T., SANTOS, C. A. B. DOS., MAGALHÃES, G. A., SAMPAIO, I. B. M., TAFURI, W.L., (2002) - Quantitative analysis of cardiac lesions in chronic canine chagasic cardiomyopathy. Rev. Instituto de Med. Tropical de Sao Paulo, 44, 5, 273-278.
3. HERMAN, V., MOGA MĂNZAT, R., RĂMNENŢU, M., (2008) – Diagnosticul în boli infecţioase comune mai multor specii de animale, Ed. Mirton Timişoara.
4. McGAVIN, M.D., CARLTON, W.W., ZACHARY, J.F., (2001) - Special veterinary pathology, Ed. Mosby, USA.
5. OLARIU-JURCA, I., (2006) – Diagnostic necropsic şi medicină legală veterinară, Ed. Brumar, Timişoara.
6. OPREAN, O.Z., (2002) – Diagnosticul necropsic la carnivorele de companie, Edit. ‘’Ion Ionescu de la Brad’’, Iasi.
7. PAUL, I., (1990) - Morfopatologia aparatelor şi sistemelor organice. Bul Inf. al SMV nr. 23-24, Bucureşti.

8. ROBINSON W. F., HUXTABLE C. R., PASS D. A., (1980) - Canine parvoviral myocarditis: A morphologic description of the natural disease. Vet Pathol. 17, 282-293.
9. ROBINSON WF, MAXIE MG., (1993) - The cardiovascular system. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals. Vol. 3. 4th ed. New York: Academic Press, 1-100.
10. VLEET, V.J.J., FERRANS,J., (2001)- Cardiovascular system, In Special veterinary pathology, Edit.by.GAVIN, M.D., CARLTON, W.W., Zachary, F.J., MOSOY, SUA Edition 3, 4, 197-333.