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Commentary

Leishmaniasis and Hypertrophic Cardiomyopathy in Dogs: A Frequent but Unknown Disease

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DESCRIPTION

Leishmaniasis is a parasitic zoonotic disease, endemic in more than 70 countries worldwide, and potentially fatal in dogs and humans. It is caused in dogs by intracellular infestation of a protozoan parasite, *Leishmania infantum* (*L. infantum*). Canine leishmaniasis causes inflammation and damage in several organs, and manifests mainly by skin, kidney and eye lesions [1]. Regarding cardiac lesions, there are few descriptions of this disease in canine cardiology, although the lesions seem to be systematic, due to pericarditis (pericardial effusion) and myocarditis (with symptoms of rhythm disturbance, weakness, syncopy, hyperthermia, dyspnea, when present) [2-5].

Cardiac lesions

Major cardiac lesions consist of lymphoplasmacytic myocarditis, detected in 100% of dogs of stage IV leishmaniasis, i.e., very severe disease, with 100% of *L. infantum* DNA detected [3]. Other lesions are described as myonecrosis, increased interstitial collagen, lepromatous-type granulomatous myocarditis, fibrinoid vascular change and vasculitis [2].

Although cardiac lesions are systematic, cardiac clinical symptoms are rarely reported, because most of infected dogs are asymptomatic. Recently a clinical case highlighted electrocardiographic and echocardiographic assessment in a female dog and identified a left ventricular arrhythmia and a significant myocardial hypertrophy, i.e., symmetric hypertrophic cardiomyopathy, with flow disturbances (turbulent flow of dynamic obstruction in the left ventricular outflow tract, secondary to the high septal thickening, and minimal mitral insufficiency). This particular phenotype of hypertrophic cardiomyopathy is described as systematic in leishmanisis-infected dogs and is precisely the consequence of the lymphoplasmacytic myocarditis described above, possibly immune-mediated [3,5].

Diagnosis and biomarkers

In this case the myocarditis was confirmed by high level of cTnI dosage (Troponin I=813.0 ng/L (U.V<25.0)). In dogs this biomarker is essential for diagnosis, and its value strongly correlates with myocardial parasite load, suggesting a direct action of the parasites on myocardial cell destruction [6]. These results are confirmed by a previous study, which indicated that the number of parasitized cells correlated with the intensity of the inflammation and with the number of granulomas [2]. Intracytoplasmic Leishmania amastigotes are found in a majority of cardiac cells and in pericardial effusion, for dogs developing chonic pericarditis [2,4]. In contrast regarding N-terminal pro B-type Natriuretic Peptide (Nt-proBNP), another cardiac biomarker used in dogs, no correlation has been found between levels of this biomarker and myocardial parasite load [7]. Additionally, authors observed a significant correlation between cTnI concentration and creatinine [3].

Challenges and future research

Because most infected dogs are asymptomatic, cardiac evaluation is not systematically included in the injury assessment of this disease. Consequently, there is little clinical data on evolution, prognosis, morbidity and mortality. Data are lacking regarding the potential evolution of the hypertrophic cardiomyopathy and the potential correlations between the importance and potential impact of the hypertrophy and improvement or aggravation of the infestation. Additionally, although a strong correlation exist between cTnI and parasite load, there is no study evaluating clinical and biological evolution of the disease in correlation to parasite load and cTnI levels.

Many questions remain unanswered to date in hypertrophic cardiomyopathy due to leishamianis in dogs, mainly because of non-systematic cardiac evaluation. Cardiac assessment should be systematic in this disease, allowing to further studies and better understanding of this particular phenotype of hypertrophic cardiomyopathy.

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