Dirofilaria spp. is mainly responsible for zoonotic infections, with *D. immitis* and *D. repens* most important species of this genus, that are simultaneously responsible zoonotic infections worldwide. Of these two, *D. immitis* is the most important from a veterinary point of view because it causes the canine and feline cardiopulmonary dirofilariasis (heartworm disease), who attended with serious diseases can cause death of the animal and from the point of view of public health as it can affect humans causing a pulmonary nodule. Heartworm disease is a cosmopolitan vector-borne disease that occurs primarily in tropical, subtropical and temperate reached their highest prevalence rates in Europe, EEUU and Far Eastern countries. Currently, there is a change in the distribution of cardiopulmonary dirofilariasis due to several factors, among which is global warming, reports of cases in countries that were considered free of this parasite. There are new models of prediction as the Geographic Information System (GIS) that have been applied to identify potentially heartworm endemic areas, annual transmission times, and forecast the situation in a very specific location.

Between 2017 and 2018, the majority of the studies carried out on the heartworm disease (a total of 174 to date) study mainly new prospects in the treatment of canine heartworm disease, macrocyclic lactone resistance, cardiopulmonary and inflammatory biomarkers, canine, feline and human seroepidemiological studies, clinical cases, in addition to systematic reviews and meta-analysis. However, there are very few studies that analyze the pathology of the disease, the origin of vascular and pulmonary damage and even, cardiac damage.

Heartworm disease usually develops a chronic progression, first showing vascular and pulmonary effects and eventually affecting the right chambers of the heart. The fundamental factors are the slow and progressive death of some worms, the mechanical damages caused by the physical presence of these in the wall of the pulmonary arteries, the immune and inflammatory response of the host against the parasites, factors excreted by worms, and against *Wolbachia* when it is released when the worms die. All of them cause structural and functional alterations of the endothelium, proliferative endoarteritis, dilatation and loss of elasticity of the arterial wall. As a result of damage in the arteries, there is later perivascular inflammation, pulmonary edema, fibrosis and pulmonary hypertension. Finally, in advanced stages of the disease, cardiac hypertrophy and congestive heart failure are observed. Parallel to this chronic development, an acute process can occur due to the mass death of adult worms in a natural way or due to the adulticide treatment used, and the release of *Wolbachia* into the bloodstream. This causes the potentiation of inflammatory reactions and the appearance of severe pulmonary thromboembolism, and also in other organs, with obstructions, impaired blood flow, hypoxia and the appearance of cardiocirculatory shock. Damage to other organs can occur, which are especially important because of their frequency and severity. They are of inflammatory origin (glomerulonephritis) and cause alteration of renal function with the appearance of proteinuria. They are mainly due to the death of microfilariae and the consequent release of *Wolbachia* in the renal tissue.

For all this, it would be necessary to promote studies on heartworm disease to better understand the pathogenesis of heartworm disease by more research groups, and better understand the source of damage to the host to be able to avoid it [1-7].

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Bibliography


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