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Canine and Feline Dirofilariasis Currently Spreads in Indonesia Damaging Pulmonary Arteries and Lungs: A Literature Review

(KAJIAN PUSTAKA: DIROFILARIASIS ANJING DAN KUCING KINI MEREBAK DI INDONESIA MERUSAK ARTERI PULMONALIS DAN PARU-PARU)

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ABSTRACT

Dirofilariasis, caused by *Dirofilaria immitis,* mostly known as heartworm disease, is an important mosquito-borne nematode zoonosis that naturally infects canids and other species such as cats, ferrets, and humans. There have been reports of heartworm infection from many countries in worldwide. Researchers have reported *D. immitis* is widely distributed in Southeast Asia because this parasitic zoonosis disease lives in temperate, tropical, and subtropical areas and can be found in colder regions. Therefore, the authors intended to provide an overview of Dirofilariasis cases in Indonesia from a global perspective. The published articles of dirofilariasis were collected and retrieved by an electronic literature search of three databases, including Google Scholar, PubMed, and Science Direct. The literature presented is intended to enhance our current understanding of the overview of *D. immitis* infection and its prevalence in Indonesia from a global perspective. *D. immitis* infection can cause caval syndrome in dogs as well as a cardiopulmonary syndrome known as Heartworm-Associated Respiratory Diseases (HARD), which can become fatal as the number of worms infecting the host increases. Whereas in cats, although the number of worms in the host are very few (one to six worms) they can cause pathological changes in the pulmonary arteries which result in a more serious infection than in dogs and endanger life.

Keywords: canine; *Dirofilaria immitis*; feline; heartworm disease; Indonesia

ABSTRAK

Dirofilariasis, yang disebabkan oleh cacing *Dirofilaria immitis,* lebih dikenal sebagai penyakit cacing jantung merupakan penyakit zoonosis yang menginfeksi kelompok anjing dan spesies lain seperti kucing, musang, dan manusia dengan nyamuk sebagai vektor perantara utama. Ada laporan infeksi cacing jantung dari banyak negara di dunia. Para peneliti telah melaporkan *D. immitis* tersebar luas di Asia Tenggara karena penyakit zoonosis ini hidup di daerah beriklim sedang, tropis, dan subtropis namun dapat ditemukan juga pada daerah yang dingin. Artikel yang dipublikasikan tentang dirofilariosis ditinjau dan dikumpulkan dengan penelusuran literatur elektronik dari tiga database termasuk *Google Scholar*, *PubMed*, dan *Science Direct*. Literatur yang disajikan ini dimaksudkan untuk meningkatkan pemahaman kita saat ini tentang gambaran infeksi *D. immitis* dan prevalensi di Indonesia dari perspektif global. Infeksi *D. immitis* dapat menyebabkan *caval síndrome* pada anjing serta sindrom kardiopulmoner yang biasa disebut dengan *Heartworm-Associated Respiratory Diseases* (HARD), dapat menjadi fatal seiring bertambahnya jumlah cacing yang menginfeksi. Sedangkan pada kucing, meskipun jumlah cacing yang menginfeksi sangat sedikit (satu hingga enam cacing) dapat

menyebabkan perubahan patologik pada arteri pulmonalis yang lebih serius daripada anjing dan membahayakan nyawa.

Kata-kata kunci: *canine*; *Dirofilaria immitis*; *feline*; Indonesia; penyakit cacing jantung

INTRODUCTION

Heartworm (HW) disease or Dirofilariasis is a zoonotic nematode infection caused by *Dirofilaria immitis* and transmitted by Culicidae. This roundworm infection is possible in numerous companion animal species, including canines and felines. This species is considered the most virulent filarial species in dogs, their definitive natural host. The adult worms cause fatal cardiopulmonary because they are large and live in the right side of the heart, leading to congestive heart failure (CHF) and coagulation disorder. Outdoor housed dogs and cats have the greatest infection risk. Simón *et al.* (2012) reported this zoonotic parasitic disease is an increasingly crucial zoonotic disease in temperate, tropical, and subtropical areas. According to the World Health Organization (WHO) roadmap 2020, the goal of eliminating dirofilariasis is expected to be achieved by 2030 (Ghasemi *et al.*, 2020).

Although canines and felines are closely related to human beings, heartworm disease is predominantly an animal health threat. This disease is generally transmitted through blood-sucking mosquitoes when they feed on hosts infected by *D. immitis*. The most significant impact on human beings may be an indirect since infection can be mistaken for more severe conditions and necessitate invasive procedures such as thoracotomy (Weese and Fulford, 2011).

Heartworm disease in felines can be lethal, but most cases are self-limiting, and numerous are asymptomatic. Veterinarians should educate clients about feline heartworm disease to make wise choices regarding heartworm prophylaxis (Litster and Atwell, 2007). The existence of *D. immitis* in dogs constitutes a chance for the human population. Within the human, the host is the causative factor of pulmonary dirofilariasis. In numerous cases, benign pulmonary nodules can at first be misidentified as malignant tumors (Morchon *et al.*, 2012).

In the year 2020, cat and dog ownership in Indonesia was estimated to total more than 20 million and only three million serveas human companions. Cat and dog ownership will increase the zoonosis risk of filarial transmission, supported by Indonesia's geography which provides a perfect environment for intermediate hosts and filarial worms. However, to date, Indonesians do not have much information on heartworm disease. As *D. immitis* is an animal health threat and zoonotic parasitic disease, this review is contemplated to enhance our current

understanding of the *D. immitis* infection overview to discover what needs doing, to answer questions, and to establish a frame of reference for a research.

RESEARCH METHODS

All relative articles have been retrieved from PubMed (https://pubmed.ncbi.nlm.nih.gov/), Science Direct (https://www.sciencedirect.com/), and Google Scholar (https://scholar.google.co.id/) database, using keywords "*Dirofilaria immitis*", "dirofilariasis", "heartworm disease", "canine", "feline", "prevalence", " and "Indonesia". We included all relevant accepted articles without publication year limitation. The main languages of article publications were English and Indonesian. Approximately, 2.781 articles were found and sorted based on the latest. As a result, there are 47 articles that can be used to support this research. There are limited articles about the prevalence of HW infection in Indonesia over the past decade, only six articles applied for this research.

DISCUSSION

Biology

Dirofilaria immitis is the roundworm, classified in phylum Nematoda; Class Cernentea; Order from Spirurida; Superfamily Filaroidea is a long slender white/grey worm measuring 15-30 cm in length with a rigid cuticle. Adult females measure 25-30 cm, while the males grow about half as long. Many worms are usually found together in a tangled mass as presented in Figure 1 (Taylor *et al.*, 2016). This filarial nematode is a mosquito-borne nematode parasite that naturally infects canids and can accidentally infect other species such as cats, ferrets, and humans. Approximately 70 mosquito species can act as intermediate hosts; *Aedes*, *Anopheles,* and *Culex* are the most common genera acting as vectors (Aiello and Moses, 2016). As humans are accidental dead-end hosts of *Dirofilaria* and not the natural hosts, in most cases, *Dirofilaria* thought that the infective larvae injected through mosquito bites perish before attaining maturity. Because natural transmittance of dirofilariasis occurs through microfilariae, which in any way does not occur in humans, dirofilariasis cannot be transmitted from person to person (Reddy, 2013).

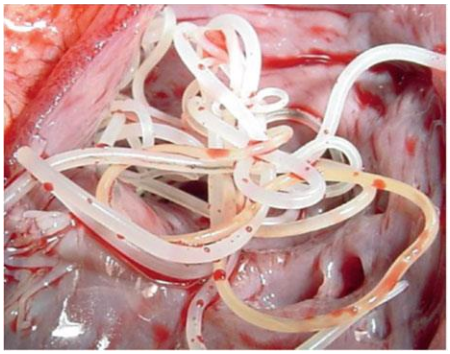

Figure 1. Adult *D. immitis* in the pulmonary artery of an infected dog (Source: Dr. Luigi Venco, In: Vismarra *et al.*, 2021)

The adults of heartworm predilect in the right ventricle and pulmonary artery of their definitive carnivore host, and pre-adults live in the pulmonary arteries. The life cycle of *D. immitis is* seven to nine months which is relatively long when compared to most parasitic nematodes (McCall *et al.*, 2008). Adult worms can live up to five to seven years in dogs, where they mate and produce microfilariae (more than 300 µm in length) circulate in the blood. An intermediate mosquito host then ingests microfilariae; this is essential for heartworm development. They migrate for an average period of around 14 days and develop to first-stage larvae (L1). Around day 10th, The L1 then moults twice and become infectious third-stage larvae (L3) that are around 1 mm in length and exist in the head of the mosquito (labium or buccal cavity). The ambient temperature must be more than 14°C for larval development in the mosquito (Bowman and Atkins, 2009). When the mosquito lands and takes a blood meal, the L3 emerges from the proboscis (namely, mosquito mouthparts) and surround the stylet (specifially, piercing part of the proboscis) in a pool of mosquito hemolymph (Fig. 2).

They then travel to the subcutaneous connective tissue and stay there for about 60-80 days, moulting into L4 larvae around day 10th and maturing into pre-adults around day 60th. These per-adults are approximately two to five cm long, and they enter the right heart via circulating blood. The pre-adults lodge in the pulmonary arteries on around day 80th and stay there for around seven weeks before returning to the ventricle by retrograde migration, becoming adults, and then mating. Some remain in the pulmonary arteries in cases of heavy infection. The prepatent period is very long, taking approximately five to six months, and sometimes longer. Any microfilariae transmitted from a bitch to her fetus via the placenta or between dogs by transfusion will continue to circulate but will not develop. Only L3 transmitted by mosquito vectors will develop into adult filariae (Beugnet *et al.*, 2018).

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Life cycle of *D. immitis* in cats similar to dogs, but microfilaremia only occurs <20% with mature HW infection and begins seven to eight months after infection. This marked reduction in worm fertility and viability is believed to result from innate resistance to *D. immitis* on feline host, where majority of immature worms are cleared, but few may continue to develop (Lee and Atkins, 2010).

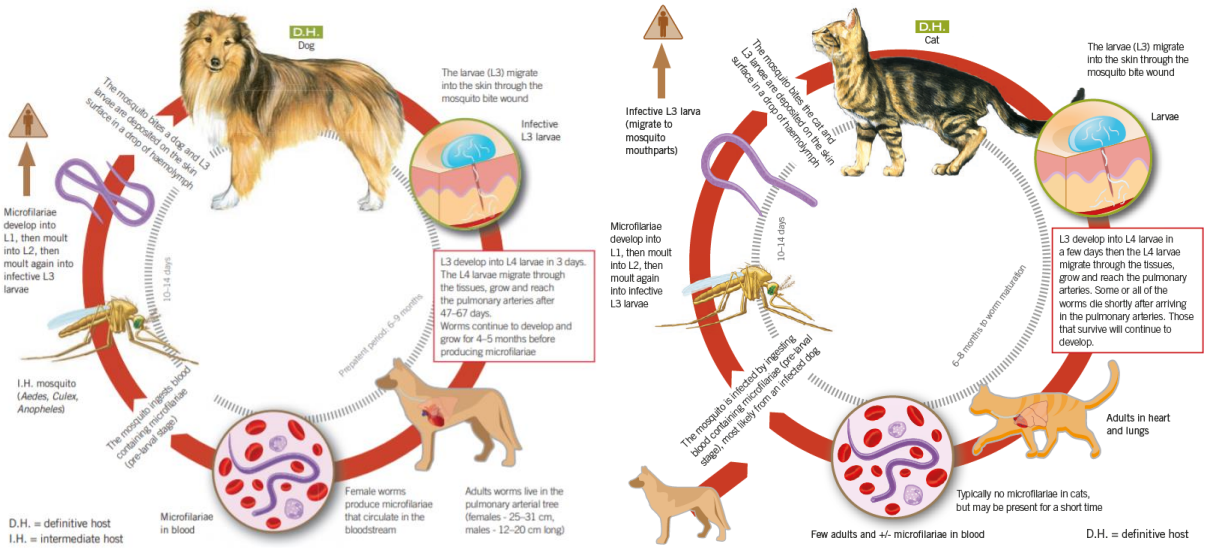

Figure 2. The life cycle of Dirofilaria immitis in the dog and cat. Microfilariae rarely found in cats due to their innate resistance to *Dirofilaria immitis*.
(Source: Beugnet *et al*, 2018)

Epidemiology

There have been reports of heartworm infection in many countries around the world. *D. immitis*, the causative agent of canine and feline heartworm disease, is widely distributed in Southeast Asia (SEA) reported in dogs and cats (Nguyen *et al.*, 2020). Because SEA is close to the equator, which has a hot climate and humidity, it is a perfect environment for them to thrive in. Mosquitoes require hot weather and suitable temperatures to produce L3 larval development in the intermediate host, as worms need temperatures greater than 14°C for nearly one month. In addition to the geographical factors (Taylor *et al.*, 2016) high density of dogs in areas where vectors are present, II) seasonal changes in the parasite and vector's development and survival rates, III) diurnal periodicity of microfilaremia (ensures that a large number of microfilariae circulate in the peripheral blood during the mosquito activity period), IV) distribution of mosquito fauna and V) age of dogs (the infection generally occurs in dogs over a year old) plays an important role in disease distribution and epidemiology. Canine Heart

Worm (Canine HW) disease occurs more often in adult dogs and large and giant breed dogs. Shorthair and rural environments are predictive for infection (Miterpáková *et al.*, 2016).

Based on meta-analysis, the prevalence of *D. immitis* in dogs in Asia was 12.07% (Anvari *et al.*, 2020). The geographical distribution *D. immmitis* infection in dogs in SEA varies in different countries, there were reports from Cambodia (Nhuong *et al.*, 2021), Myanmar (Aung, 2014; Bawm and Htun, 2015), Philippines (Theis *et al.*, 2008), Malaysia (Lau *et al.*, 2017), Singapore, Vietnam (Colella *et al.*, 2020), Thailand (Boonyapakorn *et al.*, 2008; Kamyingkird *et al.*, 2017; Kaikuntod *et al.*, 2020) and Indonesia (Erawan *et al.*, 2017). Heartworm infection in cats is still a rare case in SEA, there are only two reports in Thailand (Kamyingkird *et al.*, 2017) and Indonesia (Colella *et al.*, 2020).

There are only a few reports of HW prevalence in Indonesia for the past decade. Prevalence of *D. immitis* infection in dogs reported in Aceh was 10% (Assady *et al.*, 2016), Jakarta, Bogor and Bali were 25-57% (Fitriawati, 2009), Yogyakarta 14.6% (Erawan *et al.*, 2017), Central and South Kalimantan 28.7% and 21% (Andiarsa *et al.*, 2018), and Gorontalo 3% (Rajulani, 2013). There were only two reports about feline heartworm disease, and it was from Central and South Kalimantan with a prevalence of 26.5% and 19.7%. However, infection in domestic cats was higher than in stray cats (Andiarsa *et al.*, 2018). Another report was from Jakarta, Bogor and Yogyakarta, with a prevalence 1.3% (Colella *et al.*, 2020).

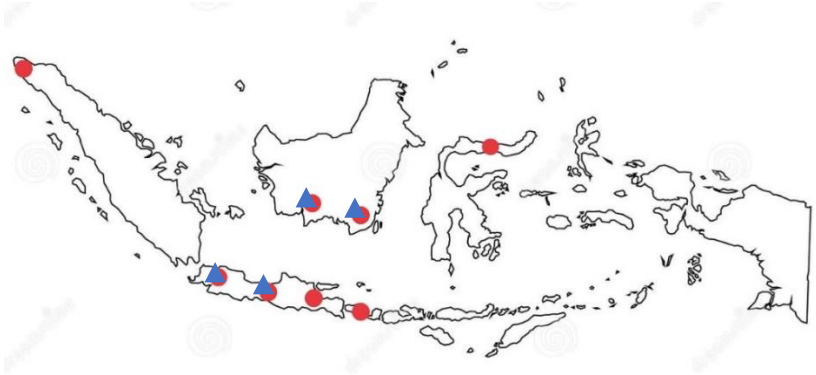

Figure 3. Map of *D. immitis* infection in dogs (circle) and cats (triangle) distribution in Indonesia.

Pathophysiology

Direct damage in the heartworm infection occurs in the pulmonary arteries and lungs. The degree of damage depends upon the number of worms present, the duration of the disease, and the host's reaction to the presence of parasites. It is believed that immature filarial causes damage when they reach the pulmonary artery three months after infection. Immature-adult

worms initiate vascular lesions and possibly pulmonary disease by causing eosinophilia and signs of respiratory illness. The adult worms typically live in the caudal pulmonary vascular, where they cause further damage through the release of vasoactive substances. The host's immunologic reaction to these substances, result in vasoconstriction and hypoxia, which leads to pulmonary hypertension and compromised cardiac output (Kitoh *et al.*, 2001). The initial vascular changes include endothelial damage and sloughing, villous proliferation, and activation and attraction of leukocytes and platelets. These changes may eventually lead to smooth muscle cell proliferation and collagen accumulation, resulting in fibrosis, causing crackles in auscultation (Atkins, 2005; Hoch and Strickland, 2008).

Death of microfilariae or presence/death of adult worms' release *Wolbachia pipientis*, an endo-symbiosis intracellular bacterium in *D. immitis* causing pneumonitis due to inflammatory reaction (Adebayo *et al.*, 2020). Haemoptysis generally implies alveolar-capillary damage or possibly parabronchial artery injury, which may be due to damage imposed by the worm itself and pulmonary hypertension. Interstitial infiltration on radiographs in dogs with HW infection presenting with a cough and dyspnoea is supportive of a diagnosis of pneumonitis. Perivascular inflammatory infiltration may result in perivascular oedema and pulmonary hypertension causes a pressure overload of the right ventricle, leading to compensatory and concentric ventricular hypertrophy. In severe cases (high worm burdens or chronic infections), chronic pulmonary hypertension with tricuspid valve insufficiency resulting in elevated cardiac filling pressure and CHF. Thromboembolism may cause acute decompensation by producing or aggravating pulmonary hypertension, right heart failure, or pulmonary infarction. Therefore, dead adult worms tend to worsen the vascular damage and enhance coagulation (Hoch and Strickland, 2008).

In severe infection, worms may be found in the vena cava posterion, causing acute hemolysis as caval syndrome (Beugnet *et al.*, 2018). A percentage of the worm burden is redistributed to the right ventricular inflow tract, leading to severe tricuspid regurgitation and decreased forward flow. Hemolytic anaemia, secondary to traumatic destruction of the erythrocytes via worm mass, also occurs. This intravascular hemolysis leads to hemoglobinuria. Some patients with caval syndrome have clinical signs of right-sided CHF (Strickland, 1998; Hoch and Strickland, 2008).

Clinical Manifestation in Canine and Feline

Most dogs with Canine HW infection are asymptomatic, regardless of worm burden and disease duration. Hoch and Strickland (2008) explained clinicopathologic manifestation of heartworm infection is in pulmonary arteries, right side of the heart, and vena cava and may include pneumonitis, pulmonary hypertension, pulmonary thromboembolism, and cor pulmonale. Clinical signs of Canine HW infection include cough, exercise intolerance, unthriftiness, dyspnea, cyanosis, hemoptysis, syncope, epistaxis, and ascites (right-side CHF), which may occur. Death of worms and thromboembolism precipitate clinical signs.

A dog may be categorized as being at low or high risk of developing clinical signs based on evaluation of potential worm burden, the health and age of the dog, and its lifestyle. There is also a more complex classification system whereby dogs are classified from I to IV based on the severity of signs. Class I dogs have a minimal clinical impairment. Class II dogs exhibit cough. Class III dogs are severely affected and variably present with cough, hemoptysis, weight loss, lethargy, exercise intolerance, dyspnea, ascites. Class IV refers to dogs with caval syndrome. Vismarra *et al.* (2021) explained "Caval Syndrome" due to a sudden increase in pulmonary pressure and the subsequent movement of worms from the pulmonary artery into the right cardiac chambers. Trouble breathing, loud heart murmur (right side of the thorax), and hemoglobinuria are pivotal clinical signs in this syndrome. Dogs aged 5-7 years are at higher risk of having a heavy worm burden, possibly due to increased exposure and for disease development.

Infected cats can be asymptomatic or present with intermittent cough, episodic dyspnea, heart failure, vomiting, lethargy, anorexia, or weight loss. Signs usually develop during two phases of the HW life cycle. Firstly, the juveniles arrive into pulmonary vasculature three to four months following infection. Secondly, adult heartworms death caused the clinical manifestation of HW disease as a cardiopulmonary syndrome called Heartworm-Associated Respiratory Disease (HARD) due to acute inflammatory reaction. Early signs are associated with a critical vascular and parenchymal inflammatory response in newly arrived juveniles and the subsequent death of many or all of these juveniles. It is frequently misdiagnosed as asthma or allergic bronchitis. The end of even one adult filarial worm can cause acute respiratory distress and shock, which may be fatal and seems to be the consequence of pulmonary thrombosis and anaphylactic-like shock (Aiello and Moses, 2016). Despite the low burden of worms (one to six adult worms per cat), severe pathological changes are seen early in cats and may be life-threatening. When immature worms enter the pulmonary vessels (about three

months post-infections), there is pulmonary hypertrophy and endo-mesoarteritis with occlusive internal hypertrophy. Pulmonary arterial disease associated with *D. immitis* is considered more severe in cats than in dogs due to the increased intravascular pulmonary macrophage activity. Caval syndrome is rarely observed in cats but may occur when one or two worms are located in the right side of the heart, causing tricuspid regurgitation (Pennisi *et al.*, 2020).

Diagnosis

The of canine and feline heartworm infection diagnostic approach should incorporate testing methods such as; immunodiagnostics, microfilariae testing, radiography, and blood analysis. Canine HW will interpret the diagnosis of heartworm disease by studying clinical tests in Table 1.

Table 1. Interpretation of heartworm diagnostic test in cats (Nelson, 2007; Litster and Atwell, 2008)

Test	Description	Result	Interpretation	Limitation
Antibody test	Serum antibodies to heartworm larvae can be detected as early as eight weeks after mosquito transmission.	Negative	Lower suspicion index	Antibodies show the infection of heartworm larvae, but do not prove that the cause of the disease.
		Positive	An increasing index of suspicion indicates that the cat is in danger.	
Antigen test	The antigen is detected in adult female filariae as well as dying male or female filariae.	Negative	Lower suspicion index	Male-only or immature worm infections are extremely rare.
		Positive	The existence of heartworms is verified.	
Thoracic radiography	Detect pulmonary artery enlargement, pulmonary parenchymal inflammation, and edema.	Normal	Lower suspicion index	Radiographic signs can be affected by clinical interpretation.
Echocardiography	Detect immature or adult heartworms in the pulmonary arterial tree lumen using echogenic walls.	Signs consistent with feline heartworm disease	The risk of suspicion is significantly increased when the arteries are enlarged.	The accuracy rate tends to be influenced by the ultrasonographer's experience with heartworm detection.
		No filaria seen	No changes to index suspicion	

Microfilariae testing. Heartworm can be diagnosed using microscopic identification of microfilariae in the blood. The diagnosis may be aided by parasitological (Buffy coat, wet mount, modified Knott's test). Microfilaria species can be distinguished using molecular tests such as the Polymerase Chain Reaction (PCR) and histochemistry.

Immunodiagnostics. Infection is ideally identified by immunodiagnostics before the onset of clinical signs; however, at the earliest, Canine HW antigenemia and microfilaremia do not appear until five and six-and-half months after infection, respectively. Diagnosis of HW using multiple blood tests to assess the presence of antigens; serological (ELISA) tests in the laboratory (Adebayo *et al.*, 2020). These tests are simple to perform, susceptible, and highly specific (Hoch and Strickland, 2008). However, since Ag serology has a low rate of false-positive outcomes, a positive result usually suggests a current infection. When there is a suspicion of heartworm infection in cats, commercially available, ELISA Ab-tests can be used as screening test (Litster and Atwell, 2008).

Radiography. Radiography is used to evaluate disease severity and assess cardiopulmonary parenchymal changes, but they are not diagnostic for heartworm infection. Radiographic changes showed enlargement of pulmonary arteries and truncation in the caudal lobar branches (Fig. 4A). Diffuse or focal bronchointerstitial parenchymal patterns are another feature but are also recognized in feline asthma and lungworm infection. On the left lateral projection, the cranial lobar pulmonary artery should not be wider than its accompanying vein or the proximal one-third of the fourth rib (Fig. 4B) (Hoch and Strickland, 2008). In feline heartworm disease, changes to structures visible on thoracic radiographs can occur less consistently than in canine heartworm disease. Echocardiography can visualize adult nematodes within the right heart or adjacent pulmonary arteries as double-lined hyperechoic structures (Litster and Atwell, 2008). The right atrium (RA) and central pulmonary artery (PA) were slightly dilated on echocardiography, heartworms are visible as double hyperechoic parallel lines (Cavaliere *et al.*, 2017). Intracardiac worm load in RA (Right atrium is assessed in several cardiac views and is estimated to be low (some worms; estimated <5) or high (more than a few in RA and filling RA) (Fig. 5) (Romano *et al.*, 2020).

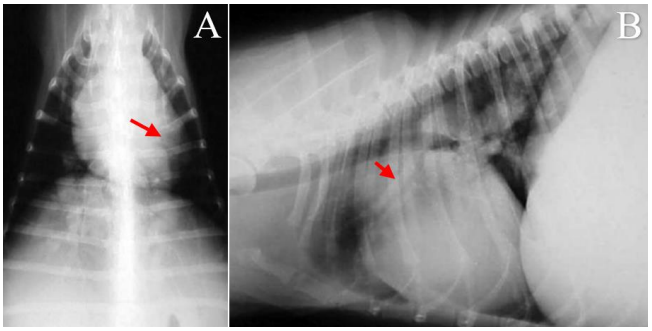

Figure 4. (A) A dog with heartworm disease has a dorsoventral radiograph that shows right-sided heart enlargement, (B) Lateral radiograph that shows significant pulmonary artery enlargement in a patient with heartworm disease (Source: Hoch and Strickland, 2008)

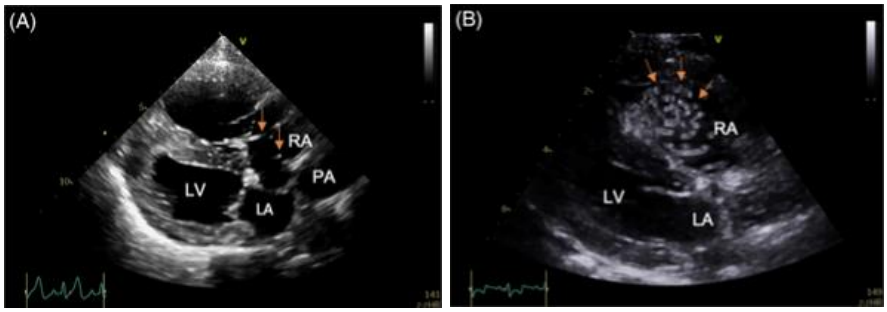

Figure 5. Echocardiography images of heart in two dogs with intracardiac heartworms that appears as parallel hyperechoic lines (arrows). (A) estimated low worm burden (<5) in the right atrium (B) estimated high worm burden in the right atrium. LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium (Source: Romano *et al.*, 2020)

Treatment and Prevention

Treatment protocols of Canine HW are anthelminthic, arsenical chemotherapeutic agent, corticosteroid, and antibiotics. The current drug used for the prevention of Canine HW is the macrocyclic lactones (MLs), which include avermectin (ivermectin, selamectin) and milbemycin (moxidectin, milbemycin oxime). As commonly administered monthly, ML does not prevent the initial infection (L3 stage) and effective in curing the infection for adult worms; it acts by killing any microfilaria then prevent them from developing into the adult, which causes disease. Adult worms can be eliminated using melarsomine dihydrochloride (melaminylthioarsenate). The American Heartworm Society (2020) recommended dose is 2.5 mg/kg IM with a 3-dose regimen, in which one injection is administrated, then two doses are given 30 days later at 24 hours intervals. It is recommended to start ML (ivermectin) preventive at the time of diagnosis or up to two to three months before melarsomine treatment (Wolstenholme *et al.*, 2015).

In one recent study of dogs with Canine HW, tetracyclines (doxycycline and minocycline) are the antibiotics of choice, but doxycycline dosed at 10 mg/kg q12h, PO for 30 days was superior to minocycline (Savadelis *et al.*, 2018; Carretón *et al.*, 2020). Half of the dosage (5mg/kg q12h) is used when gastrointestinal signs develop (Kramer and Genchi, 2014; Ames *et al.*, 2020). It is essential to eliminate *Wolbachia* in *D. immitis* because they are released due to the death of worms, and it can reduce the inflammatory side effects (Carretón *et al.*, 2020). Corticosteroid choice for HW is prednisone (0.5 mg/kg q12h for the 1st week, q24h 2nd week. And q48h 3rd week), and it is recommended to taper it concurrent with the first and third injection of melarsomine (AHS, 2020).

The incidence of symptomatic canine pulmonary thromboembolism (Canine HW-PTE) in non-arsenical adulticide protocols is unknown. The timing of Canine HW-PTE appears to be unpredictable, sometimes occurring months after initiation of macrocyclic lactone therapy (Ames *et al.*, 2020). Therapy for Canine HW-PTE usually consists of supportive care and individually tailored acute and chronic pharmacotherapy. Treatment for severely affected dogs includes strict cage rest, oxygen administration via oxygen cage or nasal insufflation (50 to 100 mL/kg/min). If pneumonitis is radiographically and clinically apparent, corticosteroids are given (Ames and Atkins, 2020).

Heartworm extraction is necessary for most animals with caval syndrome (Pariaut *et al.*, 2020). Worm extraction via jugular venotomy is required for palliation. Premedication with diphenhydramine (2 mg/kg, IM) and dexamethasone (0.2 mg/kg, IV) may reduce acute hypersensitivity reactions to worm cuticle be torn. Transthoracic or transoesophageal ultrasound can be used to visualize worms in the heart and locate the retrieval device within the heart. When using the retrieval equipment to grasp or ensnare, excessive force must be avoided, as this can lead to worm laceration and release of antigen and subsequent anaphylaxis. Transoesophageal ultrasound should also be used with care to prevent snaring or grasping too many worms at once. Once there are several negative passes or transthoracic or transoesophageal echocardiography, and a confirmation of a successful reduction of worms, the venotomy is ligated or repaired, and the skin incision is routinely closed (Ames and Atkins, 2020).

CONCLUSION

From this review, it is concluded that dirofilariasis is a widespread disease in companion animals in worldwide and causes direct damage in the pulmonary arteries and

lungs. The prevalence rate of dirofilariasis is comparatively more in temperate, tropical, and subtropical areas. Further epidemiologic studies are needed to provide more information about this emerging zoonotic parasitic disease in Indonesia.

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