Case Report

HEARTWORM EXTRACTION IN A 5-YEAR-OLD MONGREL BITCH PRESENTED WITH RIGHT-SIDED HEART FAILURE

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SUMMARY

A 5-year-old female mongrel dog was presented due to the complaint of dyspnoea and ascites. Diagnostic work-ups consisting of thoracic and abdominal radiography, abdominocentesis and fluid analysis, haematology, serum biochemistry, heartworm antigen test, electrocardiography, and echocardiography revealed right-sided congestive heart failure due to caval syndrome, atrial tachycardia, and pulmonary hypertension. After a few days of stabilization, jugular catheterization was performed under general anaesthesia and seven heartworms were isolated. The dog also received heartworm treatment in accordance to the guidelines by American Heartworm Society and other medications to control the right-sided heart failure.

Keywords: Caval syndrome, heartworm disease, jugular catheterization, pulmonary hypertension, right-sided congestive heart failure.

INTRODUCTION

Canine heartworm (*Dirofilaria immitis*) is a vectorborne, parasitic filarial nematode which resides in the pulmonary arteries. In caval syndrome, the altered right heart hemodynamic (Kitagawa, *et. al.*, 1987) and the heavy worm burden result in retrograde migration of the worms to the right ventricles, right atrium, and vena cava (Jones, 2015). Subsequently, the heartworms partially or completely occlude the closure of tricuspid valves, causing lifethreatening right-sided heart failure (Jones, 2015). Given the grave prognosis in the affected dogs and the lack of expertise, many veterinarians in Malaysia do not consider heartworm retrieval as a treatment option. This case report describes successful medical and surgical management of caval syndrome and the associated comorbidities in a dog.

CASE REPORT

A 5-year-old, female mongrel dog weighing 22.5 kg was presented due to severe abdominal distension, inappetance, and reduced activity level. The dog was not vaccinated or dewormed, and fed with home-cooked leftovers. On physical examination, the dog was alert but emaciated (body condition score: 2/5), tachypneic (respiratory rate: 60 breaths per minute), and recumbent. Auscultation revealed irregular rapid heart rate (200 beats per minute) with grade IV/VI bilateral systolic murmurs. Fluid waves were also palpable during abdominal palpation.

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Editorial history: Paper received: January 2021 Accepted for publication: April 2022 Issue Online: June 2022 Right lateral chest radiography (Figure 1a) showed cardiomegaly (vertebral heart score: 11.5), pulmonary vessel enlargement, and broncho-interstitial patterns. On the dorsoventral view (Figure 1b), there were enlarged pulmonary trunk and patchy pulmonary infiltrates. The abdominal radiography (Figure 1c) showed a loss of serosal details due to ascites.

The vertebral heart score was 11.5. The lung fields showed mixed bronchial, interstitial, and vascular patterns (Figure 1b). Dorsoventral chest radiography showing an enlarged cardiac silhouette. The pulmonary trunk and the right heart were enlarged. Tortuous pulmonary vessels were noticeable on the left thoracic region (arrows). There were also bronchial and interstitial patterns (Figure 1c). Right lateral abdominal radiography showing ascites, marked by a loss of the serosal details and a distended abdomen.

A total of 4.7 L of odourless, clear straw-coloured modified transudate (specific gravity: 1.028; protein concentration: 3.6 g/dL) was aspirated from abdominocentesis, and the re-measured body weight was 18 kg. Haematology (Table 1) showed moderate nonregenerative microcytic normochromic anaemia, mild thrombocytosis, and mild neutrophilic leucocytosis while serum biochemistry did not reveal remarkable findings, therefore ascites due to hypoproteinaemia and liver disease was ruled out. The dog also tested positive for heartworm antigen but negative for antibodies against Anaplasma phagocytophilum, Anaplasma platys, Ehrlichia canis, Ehrlichia ewingii, and Borrelia burgdorferi using a Snap 4Dx Plus test (IDEXX).

Electrocardiography (Figure 2) showed a gradual decrease and increase in heart rate of 219 bpm. There were presence of ectopic P waves and reduced QRS amplitudes (R wave: 0.7 mV), which indicate atrial tachycardia. Although mean electrical axis was within normal limit (82°), right ventricular enlargement was suspected given the presence of S waves in leads I, II, III, and aVF.

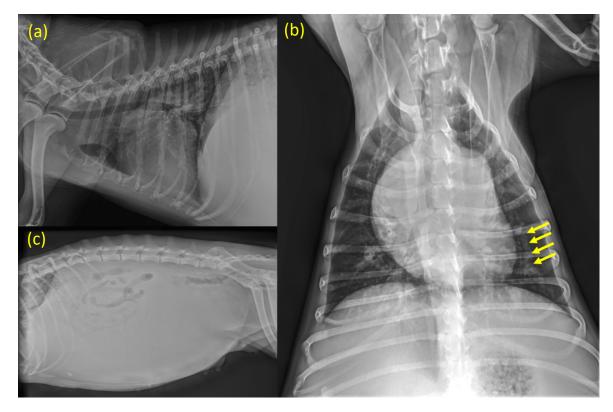


Figure 1. (a) Right lateral chest radiography showing an enlarged cardiac silhouette.

Parameter (Unit)	Value	Reference
Red blood cell (×10 ¹² /L)	4.19	5.65-8.87
Packed cell volume (%)	22.8	37.3-61.7
Haemoglobin (g/dL)	8.6	13.1-20.5
Mean corpuscular volume (fL)	54.4	61.6-73.5
Mean corpuscular haemoglobin concentration (g/dL)	20.5	21.2-25.9
Reticulocytes (K/µL)	73.3	10.0-110.0
White cell count ($\times 10^{9}/L$)	20.14	5.05-16.76
Neutrophils ($\times 10^9/L$)	16.17	2.95-11.64
Lymphocytes ($\times 10^{9}/L$)	2.61	1.05-5.10
Monocytes ($\times 10^9/L$)	1.12	0.16-1.12
Eosinophils $(\times 10^{9}/L)$	0.21	0.06-1.23
Platelet $(K/\mu L)$	485	148-484
Plateletcrit (%)	0.48	0.14-0.46
Glucose (mmol/L)	6.97	4.11-7.95
Urea (mmol/L)	4.1	2.5-9.6
Creatinine (µmol/L)	61	44-159
Total protein (g/L)	62	52-82
Albumin (g/L)	25	23-40
Globulin (g/L)	37	25-45
Alanine transferase (U/L)	29	10-125
Alkaline phosphatase (U/L)	16	23-212
Total bilirubin (µmol/L)	4	0-15
Cholesterol (mmol/L)	3.43	2.84-8.26

Note: Highlighted values represent abnormal values

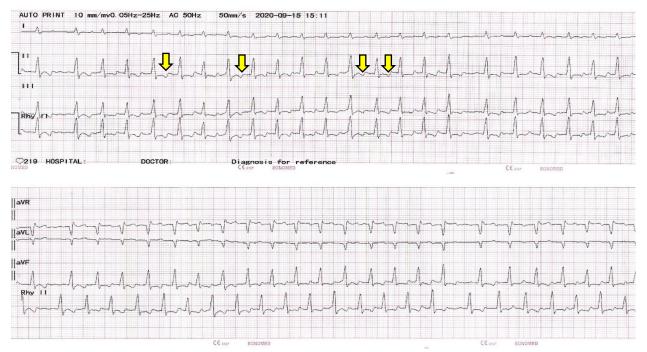


Figure 2. Electrocardiography showing a high ventricular rate of 219 bpm. Some P waves superimposed on the T waves (arrows). Right ventricular enlargement was indicated by the presence of S waves in the leads I, II, III, and aVF.

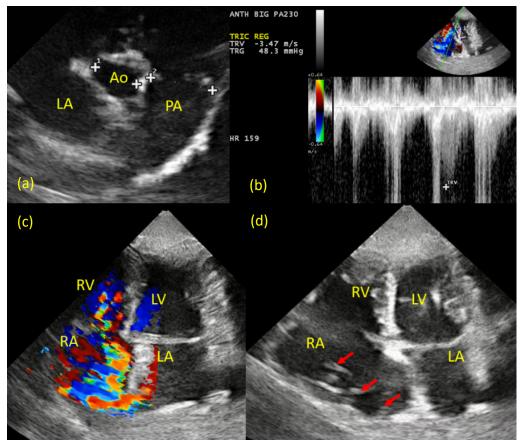


Figure 3. Findings of echocardiography; (a) the pulmonary artery **(PA)** dilated was compared to the aortic root (Ao). (b) Tricuspid regurgitation velocity was measured 3.47 m/s, giving rise to transtricuspid pressure gradient of 48.3 mmHg (c) Colour Doppler showed severe tricuspid regurgitation into the dilated right atrium (RA). (d) Multiple heartworms appeared as hyperechoic lines (red arrows) in the right atrium.

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On the echocardiography (Figure 3), dilatations of the left (left atrium to aorta ratio: 1.76) and right atria and pulmonary artery (pulmonary artery to aorta ratio: 1.41) were observed (Figure 3a). Septal flattening and paradoxical movement, as well as mitral, tricuspid (3.47 m/s), and pulmonic (2.53 m/s) regurgitations were also demonstrated (Figure 3b, 3c). These findings suggested that the dog had a high probability of pulmonary hypertension (systolic pulmonary arterial pressure: 48.2 mmHg; diastolic pulmonary arterial pressure: 25.6 mmHg), in accordance to American College Veterinary Internal Medicine (ACVIM) consensus statement guidelines. Moreover, multiple hyperechoic parallel lines resembling heartworms were also noticed in the right atrium, right ventricle, and pulmonary artery, confirming a diagnosis of caval syndrome (Figure 3d).

The dog was stabilised and treated with furosemide (Rasitol 40 mg, 1.3 mg/kg, PO, BID) to reduce the ascites, sildenafil citrate (IQNYDE 100 mg, 1.4 mg/kg, PO, BID) to treat the pulmonary hypertension, and diltiazem hydrochloride (Herbesser 30 mg, 1.7 mg/kg, PO, BID) to reduce the ventricular rates caused by atrial tachycardia. As for the heartworm disease, ivermectin and pyrantel (Heartgard, 1 tab, PO), doxycycline (200 mg, 5 mg/kg, PO, BID), and prednisolone (5 mg, 0.5 mg/kg, PO, BID) were given.

Three days later, heartworm extraction procedure was carried out. After pre-oxygenated at 5 L/min for 5 minutes, the dog was pre-medicated with fentanyl (Talgesic, 5 µg/kg, IV) and midazolam (DOMI, 0.2 mg/kg, IV). The dog was also given enrofloxacin (Baytril[®], 5 mg/kg, intravenously) as a prophylactic antibiotic, enoxaparin (Clexane[®], 0.8 mg/kg, SC) as an anti-coagulant to prevent thromboembolism, and chlorpheniramine maleate (Pirimat, 0.5 mg/kg, IV) to prevent histamine release and hence anaphylaxis due to the manipulation of the heartworms. After induced with

propofol (Troypofol[®], 6 mg/kg, IV), the right jugular area was surgically prepped. Skin and underlying tissue was carefully incised to isolate the right jugular vein. The proximal part was ligated with 2/0 nylon, followed by a nick incision distal to the proximal ligation. An angiography catheter (KDL, 5 Fr) with 4 nylon sutures tied on it was inserted via the incised vein (Figure 4a, 4b) into the right heart.

Echocardiography was performed simultaneously to visualize the heart chambers and the catheter (Figure 5a). Then, the catheter was twisted to entangle worms and pulled out slowly. The process was repeated until no heartworms were visible in the right atrium and right ventricle on the echocardiography (Figure 5b). The process successfully removed a total of 7 worms. Next, another angiography catheter was inserted into the right ventricle to measure right ventricular pressures (systolic: 48.3 mmHg; diastolic: 10 mmHg; mean: 22.7 mmHg). After that, the right jugular vein was ligated distally, followed by routine closure and bandage of surgical site. The dog recovered from anaesthesia uneventfully. Post-operatively, the dog received antiinflammatory dexamethasone (Decan, 0.25 mg/kg, IV) and analgesic morphine (0.05 mg/kg, SC). Finally, a total of 150 mL of whole blood was also transfused to replace the blood loss from the procedure.

The dog continued to receive the adulticide treatment (3-dose melarsomine protocol) in accordance to American Heartworm Society (AHS) guidelines. Additionally, medications which included pimobendan (Cardisure[®] 10 mg, 0.3 mg/kg, PO, BID), sildenafil citrate (IQNYDE 100 mg, 1.3 mg/kg, PO, BID), diltiazem (Herbesser[®] 30 mg, 1.5 mg/kg, PO, BID) were also prescribed. The furosemide dosage was tapered to 0.6 mg/kg, PO, BID before discontinued.

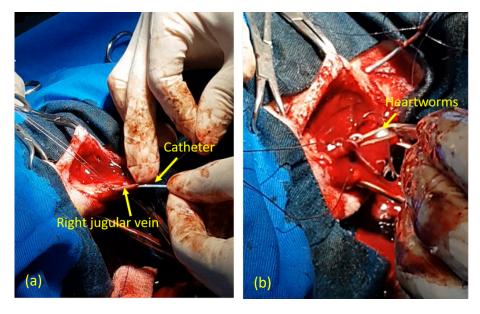


Figure 4. (a) A 5 Fr angiography catheter was inserted via the exteriorized right jugular vein into the right atrium. (b) The catheter was twisted to snare and remove heartworms.

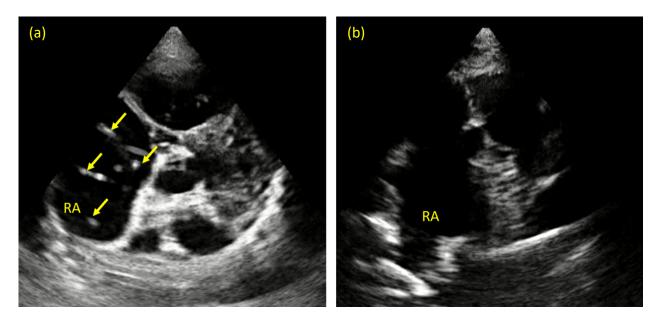


Figure 5. Echocardiography showing (a) presence of heartworms in the right atrium before the procedure. (b) After the extraction, no heartworms were visible on the echocardiography.

Several follow-up examinations showed that the dog was bright and alert with significantly reduced ascites. On day 94, the melarsomine (Immiticide[®], Merial) treatment was completed. The dog's ventricular rates were maintained satisfactorily around 157 bpm (Figure 6a). However, the echocardiography (Figure 6b, 6c) revealed an aggravated pulmonary hypertension (systolic: 70.8 mmHg; diastolic: 29.8 mmHg), so the sildenafil dosage was increased (IQNYDE 100 mg, 3 mg/kg, PO, BID). Besides, the owner was educated to provide timely heartworm preventive medicine to the dog. At present, the dog is active and eating well.

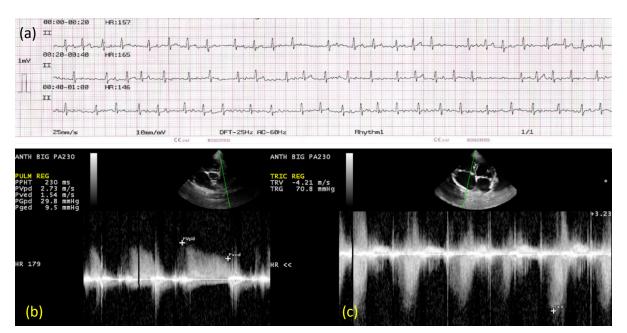


Figure 6. On day 94, the dog had (a) an average heart rate of 157 bpm despite the presence of ectopic P waves on the electrocardiography, but worsened pulmonary hypertension given increased (b) diastolic pulmonary arterial pressure of 29.8 mmHg and (c) systolic pulmonary arterial pressure of 70.8 mmHg.

DISCUSSION

Caval syndrome occurs when there is a high worm burden of more than 40-60 heartworms on average, which consequently obstructs the right cardiac hemodynamic (Atkins, 2005). On the other hand, Kitagawa et al., (1987) reported that the retrograde heartworm migration is caused by decreases in blood flow volume and velocity in the right heart, but not solely due to the high worm burdens. To the best of our knowledge, we reported Malaysia's first case of successful heartworm extraction in a mongrel dog with caval syndrome which presented right-sided congestive heart failure, pulmonary hypertension, and atrial tachycardia, the procedure was performed successfully without complications. In the present case, the low number of heartworms in the dog led us believe that atrial tachycardia and pulmonary hypertension might play a role in reducing the right cardiac output and causing the heartworm migration from the pulmonary arteries. In addition, the lower worm load resulted in milder physical obstruction to the erythrocytes, and this might explain the absence of haemoglobulinemia and haemoglobulinuria.

Given the poor prognosis of the dog if heartworm extraction was not pursued, jugular catheterization was performed to remove the heartworms from the right atrium and right ventricle immediately. A snare was made from an angiography catheter in favour of its rigid property and ability to measurement right cardiac haemodynamic, although the use of red rubber feeding tube, basket retrieval device, and alligator forceps is also recommended (Jones, 2016).

Interestingly, the invasive measurement of the right ventricular pressure corresponded to the echocardiographic estimation of systolic pulmonary artery pressure and confirmed the diagnosis of pulmonary hypertension. In the present case, the pulmonary hypertension was classified as group 5 due to dirofilariasis, based on the proposed classification of pulmonary hypertension in the dogs by the ACVIM consensus statement guidelines. After the surgical extraction, the dog also received appropriate medical treatment in accordance to the AHS guideline to eliminate the possible remaining adult worms and microfilariae.

Despite the worsened pulmonary hypertension which was believed to be due to pulmonary thromboembolism from the dead worms (Hirano *et al.*, 1992), the dog showed resolved ascites and an increased exercise level at the present, implying favourable outcomes to the current treatments with diltiazem, sildenafil, and pimobendan. However, given the significant cardiopulmonary remodeling marked by severe right cardiomegaly and pulmonary hypertension, the dog has a fair-poor prognosis in which regular veterinary revisits are warranted to monitor the disease progression.

CONCLUSION

Canine caval syndrome may be presented with rightsided congestive heart failure in the absence of haemoglobulinemia and haemoglobulinuria, and is diagnosed by echocardiography and heartworm antigen test. Heartworm extraction should be considered as a life-saving treatment option regardless of the compromised hemodynamic in the affected dogs. Post-operative medical treatments should also be given to treat or control other comorbidities.

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