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Case Report

Pericardial Effusion in a Dog with Pericardial Hemangiosarcoma

T. Gunasekaran, B.V.Sc & A.H[a], N.B. Olivier, DVM, PhD[a], R.C. Smedley, DVM, MS[b], R.A. Sanders, DVM, MS[a,*]

[a] *College of Veterinary Medicine, Small Animal Clinical Sciences, Michigan State University, 736 Wilson Road, East Lansing, MI, USA 48824*
[b] *College of Veterinary Medicine, Veterinary Diagnostic Laboratory, Michigan State University, 4125 Beaumont Rd, Lansing, MI, USA 48910*

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Abstract An adult Jack Russel terrier dog presented for evaluation of large-volume peritoneal and pleural effusion. Echocardiography revealed scant pericardial effusion and abnormally thickened pericardium. Electrocardiography revealed complete atrioventricular block with junctional and ventricular escape beats and occasional ventricular premature complexes. Computed tomography of the thorax confirmed diffuse abnormal thickening of the pericardium, and a tentative diagnosis of constrictive-effusive pericarditis was made. The dog underwent subtotal pericardiectomy to remove the parietal pericardium and permanent epicardial pacemaker implantation to manage bradycardia. Based on pericardial histopathology and immunohistochemistry, a diagnosis of pericardial hemangiosarcoma was made. Systemic chemotherapy was initiated with doxorubicin 1 month after surgery. Despite initial improvement with chemotherapy, the dog was euthanized 4 months after surgery because of development of recurrent pleural effusion. To the author's knowledge, this is the first case report in dogs to describe isolated pericardial location of hemangiosarcoma resulting in constrictive-effusive pericarditis.

* Corresponding author.
E-mail address: ras@cvm.msu.edu (R.A. Sanders).

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Abbreviations

AV	atrioventricular
bpm	beats per minute
CT	computed tomography
ECG	electrocardiography
HSA	hemangiosarcoma

A 4-year-old, 7.4-kg, female spayed, Jack Russel terrier dog presented to the College of Veterinary Medicine, Veterinary Teaching Hospital, Michigan State University, for further evaluation of bicavitary effusion that was diagnosed by the referring veterinarian. Before presentation, the dog had been coughing, intermittently tachypnic, lethargic, and hyporexic for approximately 1 month duration. Referral radiographs provided evidence of pleural and abdominal effusion. The dog was not receiving any medications other than monthly heartworm preventive at the time of presentation. On initial physical examination, the dog was quiet, alert, responsive, mildly tachypnic (respiratory rate: 42 breaths per minute), and had bilaterally muffled lung sounds. The abdomen was distended with a ballotable fluid wave. Jugular venous distension was also evident. A grade III/VI left apical, holosystolic murmur and bradycardia (heart rate approximately 60 beats per minute [bpm]) with moderately strong, synchronous pulses were identified on cardiac evaluation. No other significant abnormalities were noted.

A 5-min electrocardiography (ECG) revealed frequent non-conducted P waves, with alternating junctional and ventricular escape beats. Rare ventricular premature complexes were also noted. There was complete atrioventricular (AV) dissociation with an atrial rate of 150 bpm and a ventricular escape rate of 40 bpm consistent with a diagnosis of complete AV block. The baseline heart rate did not change after atropine response test (0.04 mg/kg, IV and recheck ECG 30 min later). Transthoracic echocardiography revealed a large amount of pleural effusion and scant pericardial effusion with no evidence of the right atrial or ventricular collapse. The parietal pericardium appeared to be thickened and hyperechoic (Fig. 1). No obvious heart base, right atrial, or proximal caval masses were noted. Mild thickening of the mitral valve was noted, consistent with mild chronic degenerative mitral valve disease. All other measured echocardiographic variables were within the normal limits. A large amount of abdominal effusion was also noted via brief ultrasonography. Complete blood count revealed mild

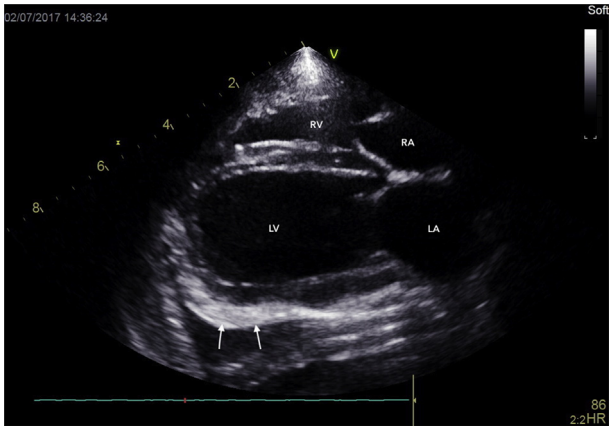

Fig. 1 Transthoracic echocardiogram—the right parasternal four-chamber view performed in a dog diagnosed with pericardial hemangiosarcoma. Note the thickened, hyperechoic pericardium (white arrows), scant pericardial effusion, and large-volume pleural effusion. LA: left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

leukocytosis (12,310/μL; reference range: 6,100—12,000/μL) that was characterized by mature neutrophilia (9,800/μL; reference range: 4,000—8,100/μL). Serum biochemistry and complete urinalysis did not show any abnormalities. To evaluate for any infectious etiology as a possible cause for third-degree AV block, serological evaluation for tick-borne diseases was performed. Interestingly, the dog was also found to have a positive titer against *Borrelia burgdorferi* (2,560; reference range: <60 = negative titer) when tested using immunofluorescent assay. The vaccination status for Lyme disease could not be confirmed from the available medical history.

At this time, given the abnormal appearance of the pericardium and the presence of tricavitary effusion, constrictive-effusive pericarditis resulting in impaired diastolic filling of the heart and systemic venous congestion was suspected. Various etiologies, including infectious pericarditis, idiopathic pericarditis, trauma, foreign body migration, and neoplasia, were considered as possible differentials. Initial treatment of the pleural effusion consisted of thoracentesis, and 0.8-L of the serosanguinous fluid was removed. Cytological analysis revealed a proteinaceous pleural effusion (4.0 g/dL), few nucleated cells (1,240/μL), and evidence for erythrophagocytosis. No infectious organisms or neoplastic cells were identified on cytological analysis. A non—ECG-gated computed tomography (CT) of the thorax and abdomen with contrast was performed to evaluate for any underlying systemic cause and to better assess the abnormal echocardiographic appearance of the pericardium. On thoracic CT

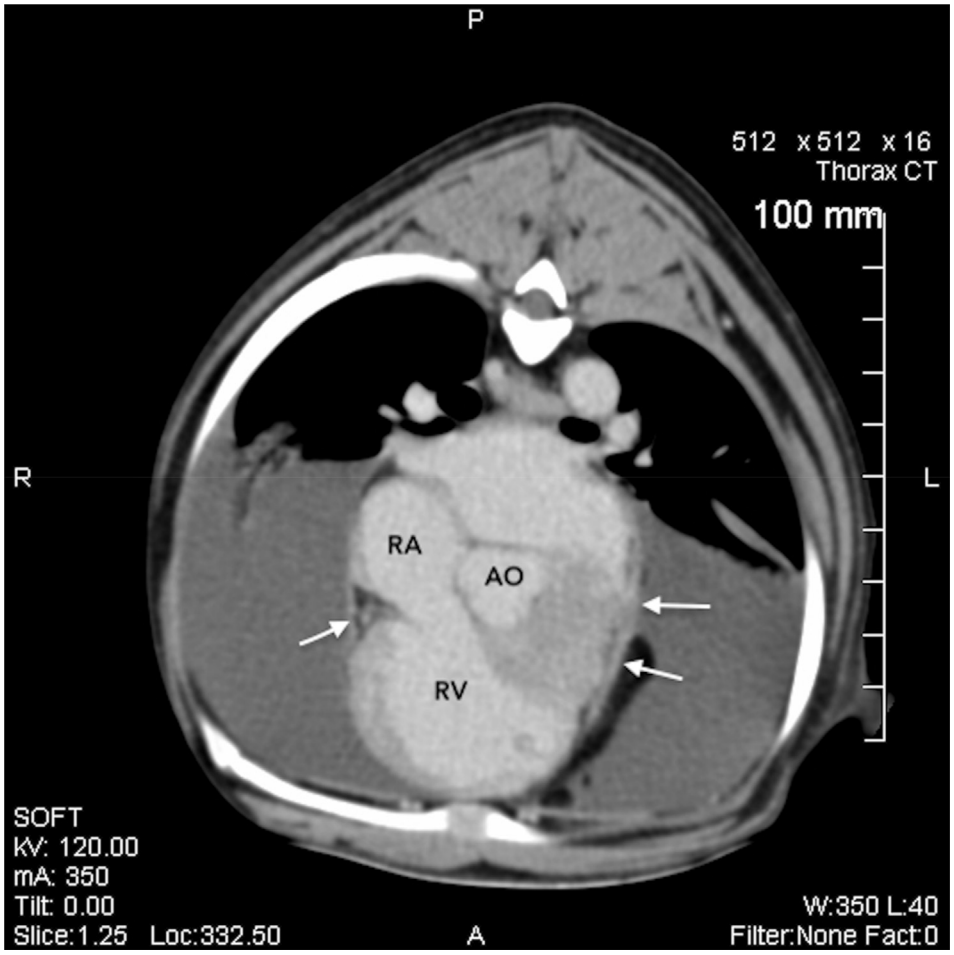

Fig. 2 Thoracic computed tomography image from a dog diagnosed with pericardial hemangiosarcoma. The pericardium (white arrows) is mildly contrast enhancing and diffusely measured 2.2 mm in thickness which could be due to inflammation, mild pericardial effusion, or neoplastic infiltration. AO, aorta; RA, right atrium; RV, right ventricle.

images (Fig. 2), the pericardium was diffusely thickened (2.2 mm) with mild postcontrast enhancement. The cranial and caudal venae cavae were mildly dilated exiting at the level of the heart base. The right ventricular free wall was thickened compared with the left ventricular free wall. Abdominal CT images revealed moderate hepatomegaly and mottling that was consistent with intrahepatic venous congestion. Incidentally, the right renal agenesis and an intrapelvic location of the bladder were also noted. Given that there was no other significant systemic pathology on advanced imaging to explain tricavitary effusion in this dog, primary constrictive-effusive pericarditis resulting in peritoneal and pleural effusion was suspected to be the cause of clinical signs. The right heart catheterization was considered for further evaluation for constrictive-pericardial disease. However, subtotal pericardiectomy via exploratory thoracotomy was elected by the owners as removal of the pericardium may alleviate the symptoms and provide histologic samples for evaluation of the pericardium. Implantation of a permanent epicardial pacemaker implantation was also recommended at the time of thoracotomy to alleviate any symptoms associated with bradycardia. Immediate pacemaker implantation was offered, but the owners elected to try therapy with doxycycline to treat for possible Lyme disease while waiting for pericardiectomy.

The dog returned 2 weeks later for pericardiectomy and epicardial pacemaker implantation. The dog was premedicated with methadone (0.3 mg/kg [13 mg/lb], IM) and midazolam (0.5 mg/kg [0.22 mg/lb], IV). After routine preparation, draping, and administration of cefazolin (22 mg/kg [10 mg/lb], IV), a temporary pacing lead[c] was positioned in the right ventricular apex

[c] Pacel™ bipolar pacing catheter, St. Jude Medical, Inc. St. Paul, Minnesota, USA.

using fluoroscopic guidance. Induction was performed with alfaxalone (2.2 mg/kg [1 mg/lb], IV), and isoflurane gas anesthesia was maintained. A right lateral thoracotomy was performed to expose the thoracic cavity. On exploration, there was diffuse thickening of the parietal pericardium. Subtotal pericardiectomy was performed with approximately 65% of the parietal pericardium removed. Samples of the pericardium for histology and tissue culture were obtained. No other significant abnormalities were noted within the thoracic cavity. A permanent steroid eluting, bipolar, epicardial pacing lead[d] was implanted in a standard fashion and connected to a pacemaker generator[e] with a baseline heart rate set at 80 bpm. Recovery from anesthesia and surgery was uneventful, and the dog was discharged from the hospital 36 h after surgery with aftercare instructions.

No bacterial growth was identified on aerobic and anaerobic cultures of the macerated pericardial tissue. The pericardial sample collected for histology during surgery was immersion fixed in neutral-buffered 10% formalin. The fixed specimen was sectioned into three pieces, routinely processed, and embedded in paraffin. Representative 5-μm sections were placed on glass slides and stained with hematoxylin and eosin for routine microscopic evaluation by a board-certified pathologist (R.C.S.) Sections of the pericardium (Fig. 3) contained a paucicellular proliferation of spindle cells that were supported by thick collagen bundles and formed irregular vascular channels containing few red blood cells, suspicious for hemangiosarcoma (HSA). Given the unusual location of the proliferation, immunohistochemical labeling for CD31 (endothelial marker) was routinely performed on the pericardial sections to confirm the diagnosis. A known positive control tissue was also routinely labeled for CD31. The spindle cells lining the irregular vascular channels showed cytoplasmic labeling for CD31 (Fig. 4), consistent with endothelial cell origin, and, consequently, a diagnosis of pericardial HSA was made. Endothelial cells lining normal blood vessels in the pericardium also showed cytoplasmic labeling for CD31 (internal control).

The dog recovered well postoperatively and returned 1 month later to the oncology service to discuss further treatment options. At that time, no concerning clinical signs were reported by the owners. Additional diagnostics were performed to aid in staging and to evaluate for any primary location of the HSA. Repeat transthoracic echocardiography revealed no pericardial effusion, mild pleural effusion, and normal global left ventricular systolic function. Complete blood count showed mild regenerative anemia (hematocrit = 40%; reference range: 42—55%) and thrombocytosis (465,000/μL; reference range: 160,000—401,000/μL). Abdominal ultrasound revealed resolution of peritoneal effusion and no

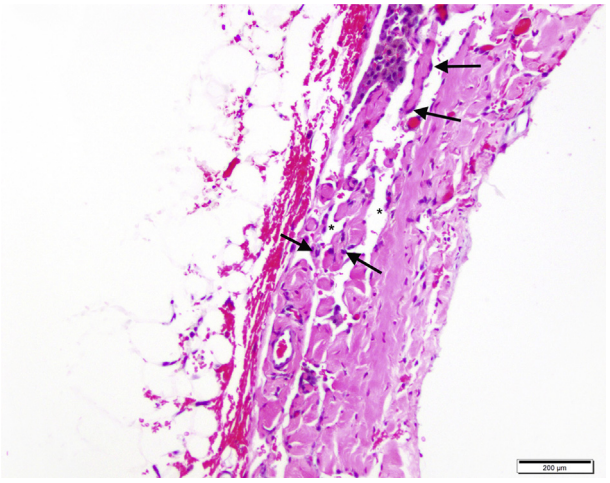

Fig. 3 Microscopically, the pericardium was expanded by a poorly cellular proliferation of well-differentiated neoplastic endothelial cells (arrows) that lined thick bundles of collagenous stroma and formed irregular channels (asterisks). Hematoxylin and eosin 10X.

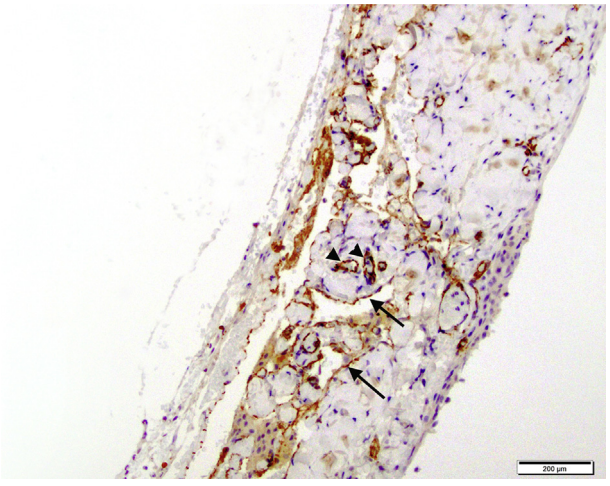

Fig. 4 The neoplastic cells showed strong dark brown cytoplasmic labeling for CD31 (arrows). The endothelial cells of normal vessels (internal control) also showed cytoplasmic labeling for CD31 (arrowheads). Diaminobenzidine chromogen, brown. 10X.

[d] Capsure® 4,698, Medtronic, Inc. Minneapolis, Minnesota, USA.

[e] Accent DR RF 2210 Pacemaker, St. Jude Medical, Inc. St. Paul, Minnesota, USA.

other abnormalities were noted. Repeat thoracic CT scan revealed an ovoid filling defect (0.9 × 0.9 × 0.8 cm) at the junction of the cranial cava and right atrium that was not previously present. This filling defect was not contrast enhancing and was suspected to be a thrombus and considered less likely to be neoplastic. There was mild pleural effusion and atelectasis of the right middle lung lobe due to chronic pleural effusion. Systemic chemotherapy with doxorubicin was initiated, and the dog received four rounds of chemotherapy (8.1 mg IV per dose) at 3-week intervals. Chemotherapy-related side-effects such as vomiting and diarrhea were managed with oral maropitant citrate (2.2 mg/kg [1 mg/lb], PO, q 24 h) and metronidazole (17 mg/kg [7.8 mg/lb], PO, q 12 h), respectively. Periodic serum biochemistry and complete blood count were performed to monitor for any significant changes. The dog tolerated chemotherapy well, and her appetite and activity level significantly improved during this time. However, 1 week after the last round of chemotherapy (4 months after surgery), the dog presented in respiratory distress due to recurrence of large-volume pleural effusion and small amount of abdominal effusion. Needle thoracocentesis of the right hemithorax was repeated and 0.6-L of serosanguinous effusion was removed. The dog initially improved after this visit, but the pleural effusion returned within 1 week after thoracocentesis. Owing to concern for the possible frequent need for thoracocentesis, despite best efforts with chemotherapy, the owners elected euthanasia. Postmortem examination was not performed at owners' request.

Discussion

Cardiac neoplasms either metastatic or primary are rare in dogs [1]. The reported prevalence of cardiac neoplasms ranges from 0.12% to 4.33% [1,2]. Most of these cardiac tumors are located in the right atrium/right appendage (63%), followed by the heart base (18%) and the left ventricle (9%) [1—6]. Of all cardiac neoplasms, HSA at the right atrium/appendage is by far the most common cardiac tumor reported in dogs [3]. In about 6.7%—25% of dogs with HSA, both splenic and right atrial masses coexist [4,5]. It is often unknown if they represent multicentric HSA or metastatic disease from one primary site [4,5]. Presumptive diagnosis of cardiac HSA is often made based on the location of the tumor [26] because needle aspiration or fluid cytology is usually nondiagnostic [6]. Cardiac tumors of pericardial origin are infrequently reported in dogs mostly as individual case reports [7]. The most commonly reported primary malignant tumor of the pericardium is mesothelioma [7]. Previously reported metastatic tumors to the pericardium in dogs include mast cell tumors, mammary carcinomas, and lymphomas [1,4]. To our knowledge, this is the first clinical case report in dogs where antemortem diagnosis of pericardial HSA is described. It is unclear if the pericardial HSA noted in this dog was the primary site of origin or a manifestation of distant metastasis. Given that there was no evidence for systemic HSA found on two separate abdominal and thoracic CT scans and no cardiac masses were visualized during subtotal pericardiectomy, primary pericardial HSA was considered as the most likely diagnosis. However, without necropsy and histological evaluation of tissues from common sites of primary HSA in dogs, a diagnosis of primary pericardial HSA could not be definitively confirmed.

This case highlights the limitations of transthoracic echocardiography and the need for advanced diagnostics in evaluating any pericardial pathology. Echocardiography has 100% specificity and 82% sensitivity for detection and characterization of masses in dogs with pericardial effusion [9]. When possible, echocardiography should be performed before pericardiocentesis as the presence of fluid provides contrast against solid masses and aids in better visualization [10,11]. However, echocardiography is not highly sensitive for the assessment of the thickness of the pericardium; as ultrasound provides a bright reflection at the pericardium—lung tissue interface, even in normal dogs, the pericardium may appear thickened and hyperreflective [9]. The presence of pleural effusion in this dog aided in better visualization of the pericardium and provided subjective assessment of the pericardial thickness. However, conclusive evidence for diffuse pericardial thickening was only noted when visually inspected at the time of exploratory surgery. Cytology of the fluid is typically not helpful in identifying the underlying cause of pericardial effusion except in cases of infectious pericarditis [8]. The diagnostic yield of fluid cytology is improved if the hematocrit is below 10% [10].

In this case, it is likely that the neoplastic disease of the pericardium resulted in constrictive-effusive pericarditis, causing poor compliance of the heart, systemic venous congestion, and recurrent effusion. In constrictive-effusive pericarditis, even after normalization of the intracardiac pressure by pericardiocentesis, elevated right atrial pressure persists [14]. Diagnosis of

constrictive-effusive pericarditis is challenging, and often the echocardiographic findings are normal [12]. Cardiac catheterization and measurement of intracardiac pressures before and after fluid administration is often required for diagnosis of constrictive-effusive pericarditis [6]. Cardiac catheterization was not performed in this dog as there was high clinical suspicion for constrictive pericardial pathology based on clinical signs. Spectral pulse wave Doppler echocardiography can be used to assess for increased respiratory variation in transmitral inflow velocities and end expiratory flow reversal in the hepatic vein in suspected cases of constrictive-effusive pericarditis [12,14]. Other echocardiographic findings consistent with constrictive pericarditis include abnormal septal (septal bounce) and posterior wall motion noted in the M-mode and a normal or increased velocity of propagation in color M-mode [12]. However, Doppler findings are not highly sensitive for constrictive-effusive pericarditis, and often a considerable amount of overlap exists between various pericardial diseases [12,14]. Cardiac CT and magnetic resonance imaging are more sensitive to detect abnormal pericardial thickness in human patients with constrictive pericardial disease [13].

Pericardiectomy was recommended for this dog as repeated thoracocentesis failed to resolve clinical signs. Traditionally, either complete or subtotal pericardiectomy is performed by open thoracic surgery. Complete pericardiectomy performed via median sternotomy has a theoretical advantage in cases of constrictive pericardial disease because it offers the best exposure and provides opportunity for complete removal of the parietal pericardium [15]. However, median sternotomy has been associated with higher morbidity compared with an intercostal thoracotomy approach [16]. Subtotal pericardiectomy via a right intercostal approach was elected in this dog, and an attempt was made to remove the majority of the pericardium. Minimally invasive procedures such as thoracoscopic pericardial window or percutaneous pericardial balloon dilation techniques would not have been suitable for this dog as they would not have provided adequate exposure for complete removal of the pericardium [17,18]. Interestingly, despite initial improvement after pericardiectomy and during chemotherapy, the dog started to develop recurrent episodes of pleural and abdominal effusion 4 months after surgery. Various possibilities need to be considered when evaluating the reasons for recurring effusion. Pericardiectomy removes only the constriction posed by the parietal pericardium and leaves the visceral pericardium intact [19]. Subtotal pericardiectomy combined with epicardial excision improved the overall outcome (postdischarge 2-year survival rate of 82%) in dogs with constrictive-effusive pericarditis due to *Coccidiomycosis sp.* infection in one study [20]. However, epicardial excision was associated with significant perioperative mortality (23.5%) in the aforementioned study [20]. Also, various complications such as coronary artery laceration, arrhythmias, and pulmonary thromboembolism are commonly reported in human patients with epicardial removal procedures [19]. Other potential causes for recurrent pleural and abdominal effusion such as pericardial constriction around the caudal vena cava, neoplastic effusion, lymphatic obstruction due to metastatic disease, or thrombosis should also be considered.

Chemotherapy with doxorubicin was recommended in this dog after pericardiectomy because surgical resection combined with doxorubicin chemotherapy increased the median survival time in dogs with cardiac HSA [21,22]. Although there was initial improvement in clinical signs, pleural effusion returned within 3 months after initiation of chemotherapy. Human patients with malignant pericardial disease require one or a combination of various therapeutic modalities, such as repeated pericardiocentesis, pericardial sclerosis, systemic chemotherapy, radiotherapy, and surgical resection [23]. Stereotactic radiation therapy was recently evaluated in dogs with right atrial HSA and was shown to reduce the frequency of pericardiocentesis [24]. However, the results of this study should be interpreted with caution because only six dogs were enrolled in the study [24].

Interestingly, the dog in this report was also diagnosed with complete AV block. Cardiac conduction disturbances including complete AV block has been previously reported in dogs with various cardiac tumors [25,26]. In addition, the dog tested positive for *B. burgdorferi* whole-cell antibodies when tested using immunofluorescent assay. However, subsequent testing to differentiate vaccine response from exposure was not performed [27]. As such, it is impossible to pinpoint the cause of AV block but various possibilities include microscopic infiltrative HSA, Lyme disease, primary degenerative conduction disorder, or systemic diseases.

In conclusion, this case report presents an unusual pericardial location of HSA in a dog. Pericardiectomy and chemotherapy improved symptoms for a short duration with eventual return of pleural effusion. Surgical removal of the visceral

pericardium might have improved overall outcome, despite risks associated with the procedure.

Conflicts of Interest Statement

None.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2019.01.008.

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