Journal of Veterinary Cardiology (2019) **23**, 122—128

Journal of Veterinary Cardiology
ESVC

www.elsevier.com/locate/jvc

Case Report

# Septic pericarditis and pericardial abscess secondary to a migrating foreign body in a dog☆,☆☆

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Received 6 July 2018; received in revised form 14 March 2019; accepted 26 March 2019

**KEYWORDS**
Epicarditis;
Canine;
Pericardiectomy;
Bacterial pericarditis

**Abstract** Abscess formation in the pericardial space has been described as a rare complication of trauma, congenital defects, penetrating foreign body, or extension of local myocardial infection in the dog. Presented here is a case of a juvenile dog with septic pericardial effusion secondary to an isolated intrapericardial abscess. Surgical pericardiectomy was successful in removing the abscess and nidus for septic effusion in this patient, and histopathology of the abscess tissue was suggestive of foreign plant material migration as the nidus for abscess formation. Recheck echocardiogram at the six-month postoperative period showed no recurrence of pericardial effusion or abscessation. Although abscess formation and septic pericarditis secondary to foreign body migration is an uncommon cause for large volume

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https://doi.org/10.1016/j.jvc.2019.03.001

pericardial effusion, this should be considered as a differential particularly in a young and previously healthy dog.


A 15.8-kg six-month-old, male, intact Weimaraner was presented to the University of Wisconsin Veterinary Care (UWVC) Cardiology Service as a referral for neutrophilic pericardial effusion and pericarditis. The patient was initially presented to his veterinarian for a three-day history of lethargy, shaking, anorexia, tachypnea, cough, and fever (103.4° Fahrenheit, 39.7° Celsius). Thoracic radiographs revealed severe globoid enlargement of the cardiac silhouette (vertebral heart scale of 13) with clear lungs. Complete blood count and chemistry panel at that time were within normal limits. The patient was referred to a local emergency and specialty center for further evaluation.

On presentation to the referral center that same day, the patient was reported to have a temperature of 100.5° Fahrenheit (38.1° Celsius). Pulse was regular at 120 beats per minute, and respiratory rate was reported at 36 breaths per minute. Heart sounds were muffled. Thoracic ultrasound was performed, and a large volume of pericardial effusion was appreciated. A coagulation profile was completed with only mild prolongation of partial thromboplastin time above normal range (prothrombin time 14 s, reference range: 11—17 s; partial thromboplastin time 128 s, reference range: 72—102 s). The patient was sedated, and 290 mL of cloudy, serosanguinous fluid was removed via pericardiocentesis. Fluid was reserved for culture and sensitivity, as well as cytologic evaluation.

Cytology of the pericardial fluid revealed total solids of 4.0 g/dL, packed cell volume of 10%, and white blood count of 235.7 K/uL. The fluid was described, as seen in Fig. 1, as containing abundant neutrophils; many of which were degenerate. Occasional macrophages and occasional reactive mesothelial cells were also described. Rare small bacilli/coccobacilli were noted within neutrophils. A diagnosis of neutrophilic exudate with rare intracellular bacteria was made. The patient was hospitalized overnight with intravenous (IV) fluid therapy and was started on oral amoxicillin-clavulanic acid (16.8 mg/kg every 12 h). He was referred to the UWVC for further evaluation the next morning.

On presentation to the UWVC, the patient was bright, alert, and responsive. The owners reported that his clinical signs had improved; he ate normally that morning, and his previous respiratory signs had resolved. His temperature on presentation was 104.2° Fahrenheit (40.1° Celsius), and his heart rate was 120 beats per minute. Femoral pulses were strong, and heart sounds were slightly muffled. Pulmonary auscultation was unremarkable. There was a 3.5-cm reducible midline umbilical hernia defect present.

Electrocardiogram at intake revealed a sinus rhythm with heart rate 140 beats per minute. The

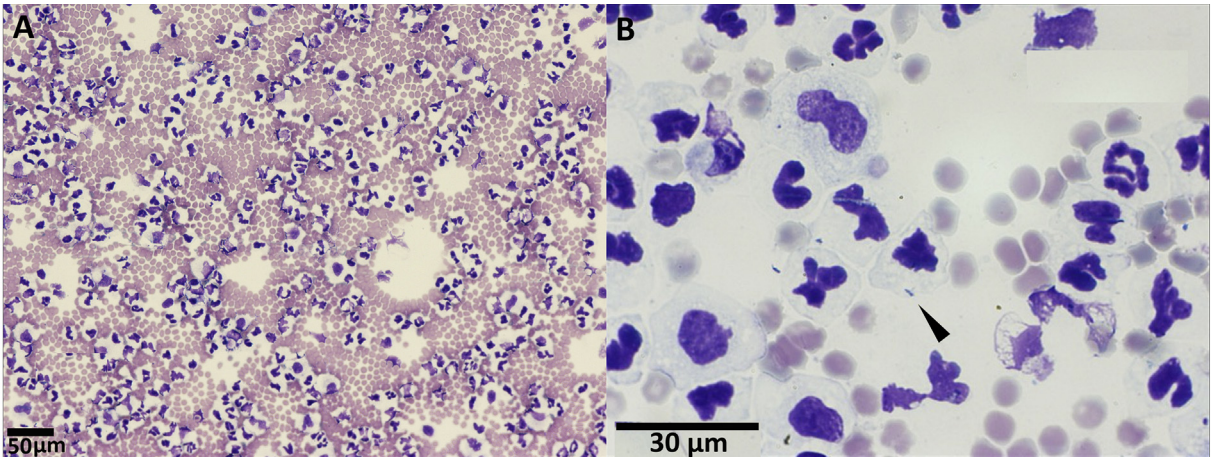


**Fig. 1** A) Sanguineous and neutrophilic pericardial exudate. 10 ×. (B) Degenerate neutrophils and intracellular bacterial bacillus (arrowhead). Wright—Giemsa stain, 100 ×.

QRS complexes were attenuated, and electrical alternans was appreciated. The patient also had a wandering pacemaker noted on the electrocardiogram. A complete blood count did not show evidence of significant leukocytosis (white blood count 13.9 K/uL, reference range: 5.0—14.0 K/uL; segmented neutrophils 8.9 K/uL, reference range: 2.6—10.0 K/uL). Peripheral packed cell volume was 40% (reference range: 40—59%), total solids 6.9 g/dL (reference range: 5.6—8.0 g/dL), and platelet count was within normal limits at 272 K/uL (reference range: 175—500 K/uL). Chemistry panel revealed mild hyponatremia (140 mmol/L, reference range: 141—150 mmol/L), mild hyperphosphatemia 8.1 mg/dL (reference range: 2.2—7.9 mg/dL), and mild hypomagnesemia 1.7 mg/dL (reference range: 1.7—2.4 mg/dL). Mild elevation was noted in aspartate aminotransferase 68 U/L (reference range: 21—53 U/L), alkaline phosphatase 172 U/L (reference range: 20—157 U/L), and creatine kinase 502 U/L (reference range: 22—491 U/L). All other values and prothrombin time/partial thromboplastin time were within normal limits. Oscillometric blood pressure evaluation was within normal limits (systolic pressure average of 143 mmHg).

Transthoracic echocardiogram on initial presentation revealed a 3.3 × 3.0 cm cystic mass fixed at the apical internal surface of the pericardium (Fig. 2A). This mass was not associated with nor affixed to the heart (Supplemental Video I). There was no evidence of blood flow through the mass on color Doppler evaluation. Moderate volume pericardial effusion with a hyperechoic and particulate appearance was noted with evidence of mild right atrial tamponade. The epicardium and pericardium had a hyperechoic appearance with a thin, shaggy layer of heteroechoic material lining the surface of each (Fig. 2B, Supplemental Video II). Pericardiocentesis was not recommended at that time given that the patient was minimally clinically and hemodynamically affected.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.jvc.2019.03.001.

The patient was admitted for hospitalization with intent for median sternotomy, subtotal pericardiectomy, and thoracic explore the following day. Intravenous fluids (Plasmalyte A) were administered at 60 mL/h. The patient was given ampicillin/sulbactam at 30 mg/kg IV every 8 h and enrofloxacin 10 mg/kg IV every 24 h. The patient was given trazodone at 4.7 mg/kg orally every 12 h as needed for anxiety in hospital. He rested comfortably and was monitored closely for hemodynamic compromise or signs of systemic inflammation or infection overnight.

The following day a thoracic computed tomography with intravenous contrast was performed which showed marked pericardial effusion. A fluid and soft tissue non-contrast—enhancing, round structure with foci of mineral attenuation and a mineral attenuating rim was noted attached to the right caudoventral aspect of the pericardial sac, consistent with the round heteroechoic mass seen on echocardiogram. A heterogenous line of fat and hepatic contrast enhancement was noted tracking from the thorax to the cranial abdomen just caudal to the diaphragm and terminating within the liver and the falciform fat. Scant pleural effusion was

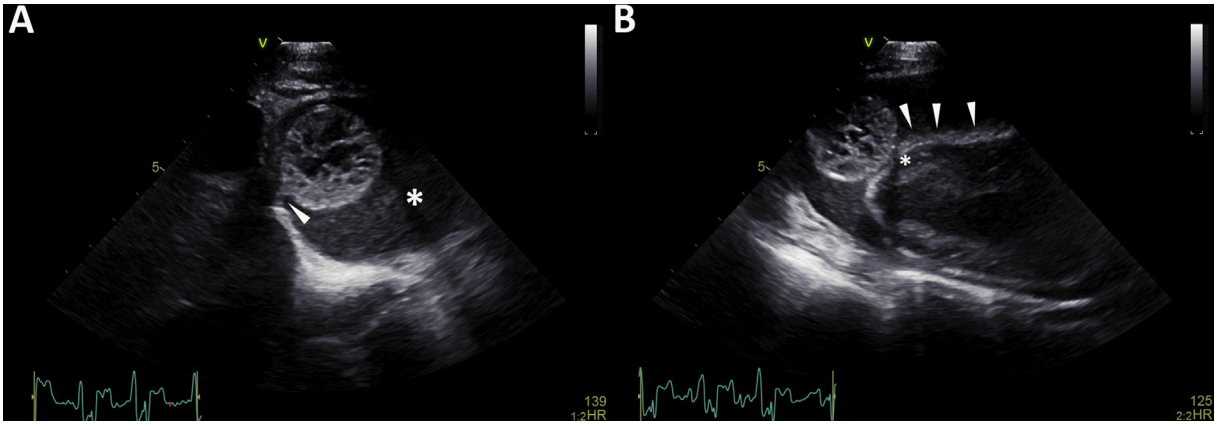


**Fig. 2** A) Obliqued apically aligned right parasternal long-axis view showing a 3.0 × 3.3 cm cavitated cystic lesion with stalk attachment (arrowhead) to apical aspect of the pericardial sac. Moderate volume of echogenic pericardial effusion present (asterisk). (B) Obliqued apical right parasternal long axis view showing cystic mass lesion adjacent, but not attached, to the left ventricular apex (asterisk). Hyperechoic and shaggy appearance is noted on the epicardial surface (arrowheads).

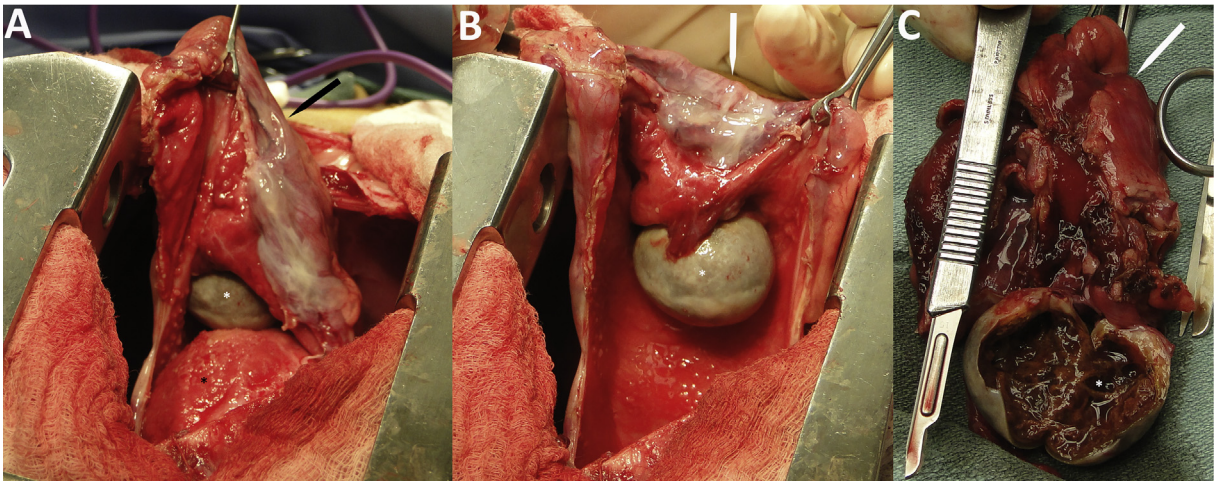


**Fig. 3** A) Intraoperative image viewing the incised thickened and fibrous pericardium (elevated with forceps-arrow) exposing the apically oriented gray-green abscess (white asterisk) and shaggy, thickened epicardial surface (black asterisk). (B) The thickened and fibrous pericardium elevated from the cardiac surface, showing fibrous stalk-like attachment of abscess to internal pericardial surface. (C) Incised abscess and associated pericardium.

noted. The patient was transported directly from computed tomography to surgery.

The patient was placed in dorsal recumbency under general anesthesia maintained with inhaled isoflurane. The thorax was clipped and prepped in a sterile fashion, and a median sternotomy was performed in standard fashion. The lungs were retracted to optimize the approach to the pericardium, which was noted to be diffusely thickened and firm. The phrenic nerve was identified and gently retracted. The pericardium was incised, and dark red, serosanguinous effusion was removed from the pericardial sac. The epicardial surface was observed to be shaggy, thickened, and irregular. The ventralmost two-thirds of the pericardial sac was removed with the assistance of a LigaSure vessel sealing device.[1] Adhered to the pericardium, adjacent to the cardiac apex, was a 2.5-cm round, firm, gray/green mass (Fig. 3). This was noted to be contiguous with a fistulous/draining tract from the mass to the subcutaneous space of the ventral thorax and diaphragm. When the region of hyperintensity and contrast enhancement in the falciform fat on preoperative imaging was evaluated, no fistulous tract or inflammation was noted caudal to the diaphragm. The mass was removed along with the associated fistulous tract and a small portion of normal diaphragm to ensure clean margins were achieved. The mass was incised once removed and green-to-brown serosanguinous fluid flowed from the cavitations within the mass. Tissue and fluid samples from the mass, pericardium, and pericardial fluid were submitted for standard aerobic and anaerobic culture. The diaphragm was closed in a simple continuous pattern, and the median sternotomy was closed in standard fashion with closure of the umbilical hernia performed concurrently. A chest tube was placed during closure through the seventh intercostal space, and negative pressure was confirmed by aspirating the chest tube after the median sternotomy was closed. The patient was recovered from general anesthesia uneventfully and was eupneic on room air at the time of extubation. Intravenous fluid therapy (Plasmalyte A) was continued postoperatively at a rate of 60 mL/h along with amoxicillin/sulbactam and enrofloxacin at the previously described doses. Pain control was maintained with a continuous rate infusion of fentanyl (3 μg/kg/hr) and ketamine (3—4 μg/kg/min). The patient rested comfortably overnight. A total of 25 mL/kg/day (473 mL total) fluid was aspirated from the chest tube in the first 24 h postoperatively.

A full echocardiogram was performed the day after median sternotomy and subtotal pericardiectomy. The remaining pericardium and epicardial surfaces were noted to be mildly irregular, hyperechoic, and thickened, consistent with pericarditis and epicarditis. The diastolic function was normal based on Doppler evaluation of mitral valve inflow velocity and tissue Doppler imaging evaluation at the level of the posterior mitral valve annulus. An electrocardiogram was performed at the time of echocardiogram and revealed a sinus tachycardia with an interventricular conduction disturbance and resolved QRS complex attenuation.

[1] Medtronic, Minneapolis, MN, USA.

The thoracostomy tube was removed once the patient's fluid production had decreased to approximately 15 mL/kg/day, on day five of hospitalization. Cytology was performed on the fluid on day two of hospitalization, which revealed a serosanguinous quality with moderate neutrophilia and no identifiable intracellular or extracellular bacteria. Although fluid production was above the standard acceptable level of fluid production, the patient's fluid production had stabilized over the preceding three days, and he was clinically feeling well. The patient was discharged from the hospital after five days of postoperative supportive care with seven days of oral amoxicillin/clavulanic acid (15.7 mg/kg every 12 h), enrofloxacin (8.7 mg/kg every 24 h), carprofen (2.2 mg/kg orally every 12 h), tramadol (3.2 mg/kg every 12 h), and a 50-μg/h fentanyl patch.

The mass and associated pericardium was submitted to the UWVC Anatomic Pathology Service for histopathologic evaluation. The histopathologic characteristics of the mass were consistent with a chronic abscess and granulation tissue, and the associated pericardium was consistent with chronic neutrophilic, lymphoplasmacytic, and granulomatous pericarditis. Small fragments of polarizable and refractile material were seen embedded in the lining of the abscess (Fig. 4). This refractile substance was suspected to be a foreign plant material. No histologically identifiable bacteria, fungal organisms, or acid-fast staining organisms were seen.

Aerobic and anaerobic cultures taken intraoperatively showed no growth from any sample after seven days of incubation, but the culture sample submitted from the time of initial pericardiocentesis revealed scant growth of *Pasteurella canis* susceptible to all antibiotics.

At recheck evaluation six months after discharge, the patient was reported to be feeling well and clinically unaffected at home. He was normally active with no collapse, weakness, or lethargy appreciated. Recheck echocardiogram allowed identification of the remaining pericardium, which was noted to be thickened, hyperechoic, and causing shadowing artifact. No epicardial echogenic abnormalities were identified in the regions of the heart without the overlying pericardium remaining. The remainder of the echocardiogram was within normal limits, with normal systolic and diastolic function. No pleural effusion was identified. A recheck complete blood count was within normal limits. No other abnormalities were identified.

## Discussion

Abscess formation in the pericardial space has been described as a rare complication of trauma,

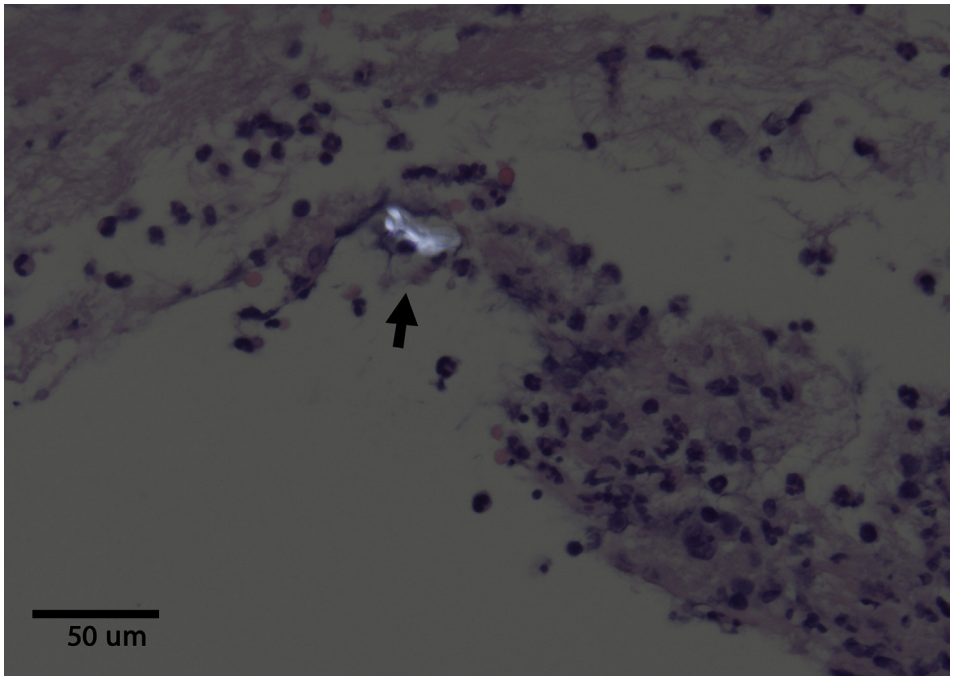


**Fig. 4** Contents of chronic abscess within pericardial sac, including fibrin, necrotic cellular debris, neutrophils, erythrocytes, and a small fragment of colorless refractile material (arrowhead). Image obtained under polarized light to highlight foreign material. Hematoxylin and eosin stain, 60×.

congenital defects, penetrating foreign body, or extension of local myocardial infection in the dog [1,2]. An abscess forms as an attempt to wall off a focus of infection or inflammation from the remainder of the body. This process is uncommonly localized to the pericardial space alone. A case reported by Parra et al. [1] detailed a similar presentation of a cavitated intrapericardial mass in an 8-month-old Greater Swiss mountain dog, with secondary serosanguinous pericardial effusion. In this case, however, a definitive diagnosis of abscessation in the pericardial space could not be made on the basis of histopathologic findings. In addition, the case lacked concurrent neutrophilic, purulent pericardial effusion to further support the diagnosis of an inflammatory and/or infectious process. After subtotal pericardiectomy and removal of the cyst-like structure, the granulomatous, cystic lesion was suspected to be a resolving abscess or hematoma. No infectious agents or fungal organisms were identified in this case.

The cavitated mass in the case presented here, when seen in conjunction with degenerative neutrophilic pericardial effusion, heightened suspicion for the presence of an abscess and secondary septic pericarditis over other differential diagnoses. Cavitated masses associated with the pericardium or myocardium in dogs have been reported with other underlying etiologies [3—5]. Such intrapericardial masses may include intrapericardial cysts, hematomas, fungal granulomas, hemangiosarcomas, lymphosarcomas, myxosarcomas, and other neoplasms. The ultrasonographic appearance of which may range from solid to cavitated, cystic, or heteroechoic on echocardiogram in the dog [4—6]. The biologic behavior of these masses is similarly variable.

Abscesses and solid masses have the potential to generate significant effusion secondary to hemorrhage, inflammation, necrosis, secondary infection, or paraneoplastic syndromes in the dog [3,7]. The presence of exudative pericardial effusion in this case was supportive of the presence of infection or inflammation as the underlying cause for the mass identified on echocardiogram. Neutrophilic or inflammatory pericardial effusions and pericarditis, however, have been reported with a variety of conditions in the veterinary literature, including systemic or local infectious etiologies (fungal, bacterial, viral) and extension of severe abscessation or infection of cardiac tissues including myocarditis or myocardial abscess in the dog [2,8—11]. Although a mixed population of small rods and coccobacilli was noted intracellularly in the effusion before antibiotic administration, only scant growth of *Pasteurella* was achieved on culture plated from initial pericardiocentesis samples. This is in contrast to the typical mixed population of bacteria cultured from previously reported cases of septic pericarditis secondary to suspect foreign body migration in a dog [7]. The lack of positive culture from intraoperative samples may be related to antibiotic administration before obtaining surgical samples in this case. For this reason, the presence of neutrophilic pericardial effusion was not able to be used as a definitively diagnostic presurgical finding for septic pericarditis and/or pericardial abscessation in this case.

The thickened and hyperechoic appearance of the epicardial surface on the postoperative echocardiogram, as well as the intraoperative appearance of a thickened and irregular epicardial surface, raises concern for constrictive epicarditis as a complication of pericarditis. Constrictive epicarditis has been reported as a component or sequela of constrictive pericarditis, even after subtotal pericardiectomy in human case reports [12—14]. Constrictive epicarditis or epicardial sclerosis shares a similar physiology to that of constrictive pericarditis, wherein diastolic filling and ventricular compliance are restricted because of a thickened and fibrous epicardial lining, developed as an accumulation of scar tissue as a response either to chronic inflammation or a resolved episode of severe inflammation or infection [14]. Human case reports cite a timecourse of weeks to months after pericardiectomy before constrictive epicarditis and diastolic impairment are identified, and it is unclear whether this information may be applied to veterinary patients with pericardial disease. Management of constrictive epicarditis in human patients may involve excoriating or cross-hatching the scarred epicardial surface to allow for more appropriate diastolic function [12]. Although the epicardial surface was irregular and thickened at the time of surgery, epicardial excoriation was not elected at the time of surgery as no diastolic impairment was identified on initial echocardiogram. It is unclear the factors that are involved in the development of constrictive epicarditis in some but not all pericarditis patients. Based on the normal appearance of the epicardium and normal systolic/diastolic function on recheck echocardiogram six months after subtotal pericardiectomy and pericardial abscess removal, however, no evidence of this complication was seen.

The case report described here is of a dog with septic pericarditis and pericardial abscess formation secondary to suspect migrating plant material. Pericardial effusion cytology and transthoracic echocardiogram were important

diagnostic features of this case. Surgical removal of the pericardial abscess and antibiotic treatment resulted in successful resolution of the abscess and septic pericarditis.

## Conflicts of interest statement

The authors do not have any conflicts of interest to disclose.

## Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2019.03.001.

Video table:

| Video | Title | Description |
|---|---|---|
| I | Two dimensional transthoracic echocardiogram of pericardial abscess | Cineloop of abscess adjacent to the left ventricular apex |
| II | Two dimensional transthoracic echocardiogram of the epicardial surface | Cineloop of right parasternal long axis view featuring shaggy, hyperechoic epicardium, moderate volume pericardial effusion, and mild cardiac tamponade |

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