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Atypical loculated pericardial effusion with infectious pericarditis mimicking a cardiac mass in a dog: a case report

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Abstract

A 13-year-old, Maltese dog presented with syncope and lethargy. Abdominal ultrasonography demonstrated anechoic peritoneal effusion and hepatic congestion. A focal echogenic round mass compressing the right ventricle and atrium was observed on echocardiography. Cardiac tamponade and right ventricular outflow tract obstruction occurred. On computed tomography, a homogeneous soft-tissue structure compressing the right chamber without contrast enhancement, suspected to be loculated pericardial effusion. During pericardiocentesis, cardiac tamponade was resolved, and irregular pericardial thickening was noted. Pericardial effusion was exudate and gram-positive bacterial colonies were observed on cytology. A diagnosis of fibrinous pericarditis secondary to bacterial infection was established.

Keywords: dogs; heart neoplasms; computed tomography; echocardiography; heart

In dogs, the most common causes of pericardial effusion include pericardial effusion secondary to cardiac tumour and idiopathic pericardial effusion [1]. A rare cause of pericardial effusion is infectious pericarditis. On echocardiography, pericardial effusion could be anechoic to echoic depending on the aetiology; however, its distribution is typically diffuse, surrounding the entire heart with a smooth margin regardless of the cause [2].

This report presents a rare case of atypical loculated pericardial effusion that formed a focal mass resulting in a severe mass effect that mimicked a cardiac tumour in a dog with infectious pericarditis. Particularly, this report elucidated the echocardiography and computed tomography (CT) features of loculated pericardial effusion in the dog with pericarditis to aid in distinguishing loculated pericardial effusion from a cardiac tumour.

A 13-year-old, 1.5-kg, spayed female, Maltese dog manifested with syncope and lethargy. On physical examination, the dog presented with cold limbs, tachycardia, and grade 4 systolic heart murmur. The body temperature was 37.2°C. Complete cell count and blood chemistry did not show any abnormalities except for anaemia (hematocrit, 24.7%; reference interval, 36.9%–55.0%).

Biatrial enlargement and caudal vena cava dilation were suspected on thoracic

radiographs (Vertebral heart score, 9.5; vertebral left atrial score, 2.9) (Fig. 1A and B). A decrease of the serosal detail was noted on abdominal radiographs. On abdominal ultrasonography, anechoic peritoneal effusion was identified. Dilation of the caudal vena cava and hepatic vein were observed. Ultrasound-guided paracentesis was performed, and fluid analysis demonstrated that the peritoneal effusion was a transudate (total nucleated cell count, 220 cells/mcL; reference interval < 1,500 cells/mcL; total protein, 2 g/dL; reference interval < 2 g/dL). Thus, right heart failure was suspected, and echocardiography was performed.

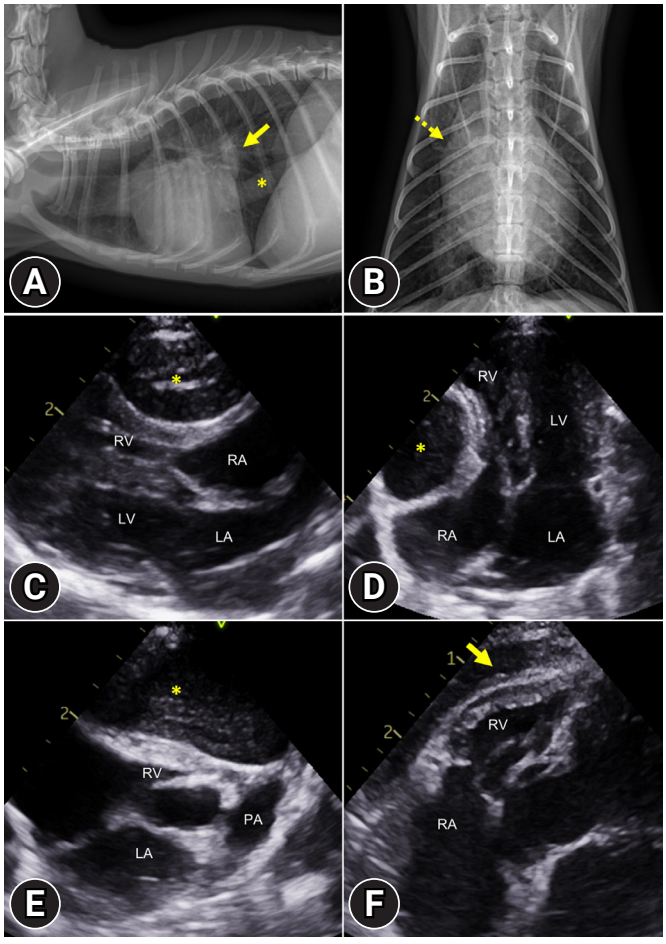

Fig. 1. Right lateral (A) and ventrodorsal (B) thoracic radiography of the dog. Left atrial enlargement (yellow arrow) and cauda vena cava dilation (yellow asterisk) are suspected on lateral image (A), and right atrial enlargement (yellow dotted arrow) is suspected on ventrodorsal image (B). Right parasternal long axis 4-chamber (C), left apical long axis 4-chamber (D) and right parasternal short axis (E) views on echocardiography. A focal echogenic round mass (yellow asterisk) lateral to the right ventricle (RV) and right atrium (RA) compressing the RA and RV (C, D) and right ventricular outflow tract (E). During pericardiocentesis (F), there is an irregular thickening of the pericardium (yellow arrow) observed on the left parasternal short axis view. PA, pulmonary artery.

A focal hypoechoic to echogenic round mass (diameter, 10.27 mm) protruding from the free wall between the right ventricle and atrium was shown on transthoracic echocardiography (Vivid E90; GE Healthcare, USA) (Fig. 1C–E). Although color-Doppler showed no blood flow signal in the mass, sensitive evaluation was difficult due to cardiac motion. Severe compression to the right atrium and ventricle was noted secondary to the mass. The right atrium could not dilate in the ventricular systolic phase (cardiac tamponade), with an increase in trans-tricuspid velocity (1.43 m/s; reference interval, 0.50–0.98 m/s). The right ventricular outflow tract (RVOT) compression secondary to the mass led to RVOT obstruction with systolic turbulent flow and increased velocity (4.47 m/s; reference interval, 0.50–1.50 m/s). Interventricular septal flattening was noted. Trans-mitral flow was decreased (E peak, 0.37 m/s; reference interval, 0.52–0.81 m/s; A peak, 0.51 m/s; reference interval, 0.45–0.78 m/s), and left ventricular diastolic dimension was reduced on M-mode (10.03 mm; reference interval, 15.3–21.3 mm). Based on echocardiography, we suspected right-side heart failure secondary to cardiac tamponade induced by the right chamber compression from the right free wall mass such as hemangiosarcoma. CT was performed to evaluate the origin and structural characteristics of the mass.

General anaesthesia was induced through an intravenous injection of 6.0 mg/kg of propofol (Anepol; Hana Pharm, Korea) and maintained using 2% isoflurane after endotracheal intubation. During anaesthesia, a ventilator was used, which was temporarily discontinued during CT scan to induce apnoea. CT images were acquired using a 16-channel multidetector CT (Alexion; Canon Medical System, Japan) with 120 kV, 150 mA, 2.0-mm slice thickness and 0.75-second rotation time. Post-contrast images were acquired after intravenous administration of 600 mgI/kg iohexol (Bonorex 300 Injection; Daehan Pharm, Korea).

Thoracic CT demonstrated a focal, homogeneously soft-tissue attenuating round structure (17.34 × 10.14 × 30.92 mm, 33 HU) at the cranioventral part of the right ventricle and atrium (Fig. 2). The structure failed to exhibit contrast enhancement except for a mild contrast-enhancing margin, connecting to the outer layer of the pericardium. Pericardial thickening was not observed. The structure led to right ventricular and atrial compression. Owing to the absence of contrast enhancement, a loculated pericardial effusion was suspected.

Ultrasound-guided pericardiocentesis was performed. During pericardiocentesis, irregular pericardial thickening was observed on echocardiography (Fig. 1F). After pericardiocentesis, cardiac tamponade resolved. Resolution of the RVOT obstruction was

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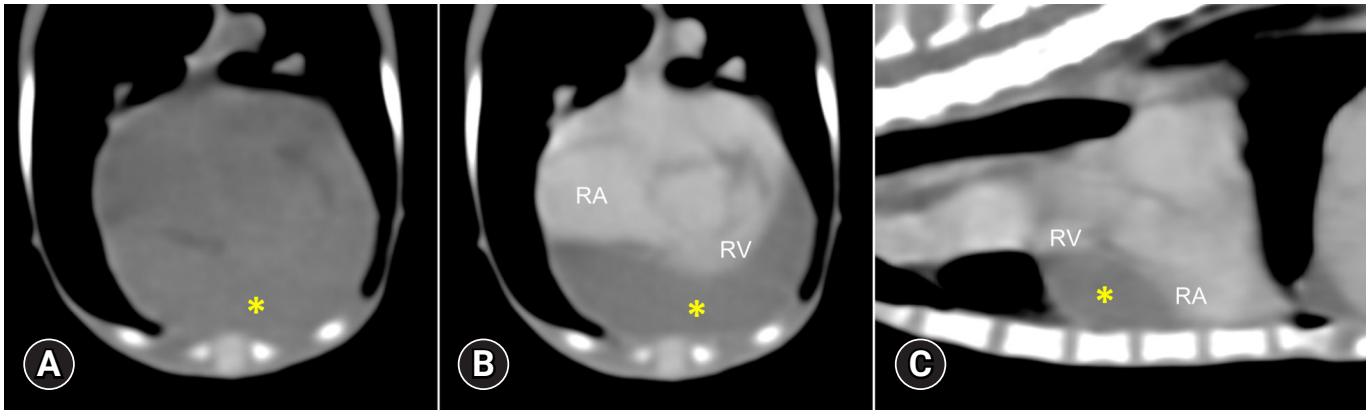

Fig. 2. Pre-contrast transverse (A) and post-contrast transverse (B) and sagittal (C) plane computed tomography images. Focal, homogeneously soft-tissue attenuating round structure (yellow asterisk) is noted at the cranioventral part of the right ventricle (RV) and right atrium (RA) compressing the right chamber. This structure does not demonstrate contrast enhancement.

also observed, with a decrease in the velocity (1.09 m/s; reference interval, 0.50–1.50 m/s). The trans-tricuspid velocity decreased to the normal range (0.79 m/s; reference interval, 0.50–0.98 m/s), indicating absence of constrictive patterns.

Grossly, pericardial effusion presented with a cloudy brown colour. On fluid analysis, pericardial effusion was confirmed as an exudate (total nucleated cell count, 9,410 cells/mcL; reference interval > 5,000 cells/mcL; total protein, 5.4 g/dL; reference interval > 2 g/dL). Necrotic and inflammatory cells admixed with fibrinous matrix was revealed using the Diff-Quik stain (Fig. 3A–C). A few neutrophils including degenerative neutrophils, macrophages, and bacteria colonies were noted. With gram stain, the number of gram-positive bacterial colonies was revealed (Fig. 3D). The final diagnosis was fibrinous pericarditis secondary to bacterial infection.

Pericardiectomy was suggested to the owner. However, the owner did not consent to surgery and antibiotics including enrofloxacin (10 mg/kg once daily, orally, Baytril Flavour Tablets 50 mg; Bayer, Germany) and metronidazole (15 mg/kg twice daily, orally, Flasinyl tab; HK inno.N Corp., Korea) were administered. A total of 9 follow-up were performed until the patient expired, with a frequency of 1 to 2 times per week for over 2 months. Loculated pericardial effusion recurred during follow-up, and pericardiocentesis was performed 5 times, approximately every 2 weeks. The dog died at home 7 days after the last follow-up (approximately 2 months after diagnosis) with acute respiratory distress. Necropsy was not conducted.

To the best of our knowledge, this is the first report of loculated pericardial effusion with infectious pericarditis in a dog. On echocardiography, the loculated pericardial effusion demonstrated as an echogenic mass within the right free wall,

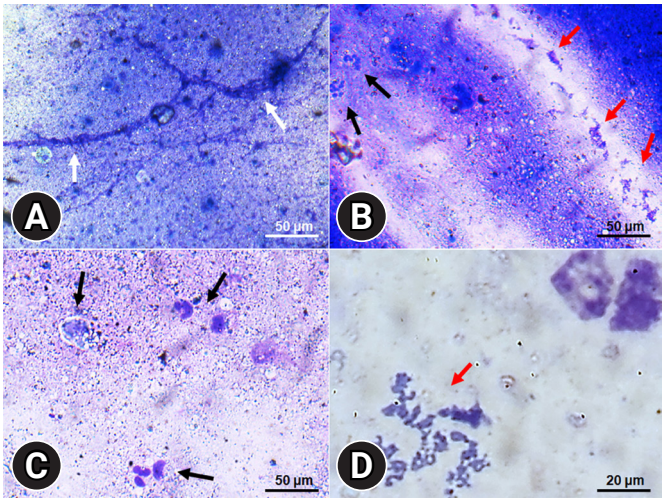

Fig. 3. Cytology using Diff-Quik stain (A-C) and gram stain (D) of the pericardial effusion. On Diff-Quik stain (A-C), necrotic cells admixed fibrinous matrix (white arrows), neutrophils including degenerative neutrophils (black arrows), and bacteria colonies (red arrows) are noted. The number of gram-positive bacterial colonies (red arrow) are observed with gram stain (D). Scale bars: (A-C) 50μm, (D) 20 μm.

leading to severe right chamber compression with a consequent cardiac tamponade and RVOT obstruction. Due to its echogenicity, rounded shape, and mass effect, it mimicked a cardiac tumour; however, based on contrast enhancement, CT distinguished it from a cardiac tumour.

In dogs, infectious pericarditis is a rare aetiology of pericardial effusion [1,2]. In a previous study, only 4.7% of dogs with pericardial effusion were diagnosed as pericarditis [1]. In the study, bacterial agents included *Bacteroides* spp., *Actinomyces* spp., *Streptococcus canis*, and *Pasturella*. Although there is a lim-

itation in the present case that a histopathologic examination was not performed, a tentative diagnosis of infectious pericarditis was possible through cytology; a large number of gram-positive bacterial colonies were identified on cytology in the current case. However, the causative bacteria were unknown due to culture failure. The rationale for the culture failure was unknown; however, possible reasons include the presence of numerous necrotic cells, scarcity of viable bacteria, and viable but non-culturable state of the bacteria.

Previously reported etiologies of infectious pericarditis in dogs and cats include foreign body migration such as grass awns or esophageal foreign bodies, trauma, administration of immunosuppressive drugs, hematogenous spread, and local extension of infectious [3]. In some cases with infectious pericarditis, the etiology of infection was not identified, and in the present case, the cause of infection was not fully determined. In our case, there were no findings suspicious for trauma, such as trauma-related history or fractures, so it was thought that trauma was unlikely to be the cause of infectious pericarditis. Although it was difficult to completely rule out small foreign bodies such as grass awns through imaging modalities, there were no findings suspicious for foreign body migration to the pericardial tissue on echocardiography and CT. The patient had taken medication for a year for seizures of unknown cause 5 years ago, and may have taken immunosuppressive drugs at that time, but had not taken any medications for 4 years before admission to our hospital. There were no symptoms such as pyrexia that would suggest systemic infection, and no obvious abnormalities in other organs were seen in radiographs, abdominal ultrasound, and CT. However, since blood and culture were not performed, which is a limitation of this case.

Only a few reports described echocardiographic features of pericarditis in dogs [4,5]. Echogenic pericardial effusion may be observed with pericardial thickening or mobile linear hyperechoic structure representing fibrins in the pericardial effusion. In the present case, pericardial effusion was echogenic and after pericardiocentesis, an irregular pericardial thickening was noted and these findings corroborates with previous reports. However, echogenic pericardial effusion may also be observed in dogs with a cardiac tumour or left atrial rupture [6]. Moreover, in chronic pericardial effusion, mild pericardial thickening could occur.

Regardless of the cause, pericardial effusion typically circumferentially surrounds the heart [2]. In the current case, loculated pericardial effusion formed a round-shaped echogenic structure with a marked mass effect leading to right atrium, ventricle and RVOT compression. These findings were atypical, which mimicked a cardiac mass. A few reported cases of loculated pericardial effusion were present in dogs; however, in all dogs, the cause of the loculated pericardial effusion was induced by trauma [7,8]. Furthermore, a detailed imaging feature of a loculated pericardial effusion is lacking in dogs.

In humans, there have been several reports of loculated pericardial effusion with infectious pericarditis [9,10]. When bacteria invade, activated leukocyte migration stimulates coagulation and subsequent fibrin formation. Over time, fibrin accumulation increases, pericardial occlusion secondary to pericardial adhesion and thickening may occur. If the absorption of effusion is insufficient, loculated pericardial effusion may arise. In our case, a significant amount of fibrin was confirmed on cytology, and pericardial occlusion was suspected to be the cause of loculated pericardial effusion.

Echocardiography is the first choice for pericardial effusion diagnosis; however, differentiation between a loculated pericardial effusion and a cardiac mass or adjacent structure may pose challenges [11]. In humans, a number of cases of loculated pericardial effusion was misdiagnosed as other structures on echocardiography, such as a cardiac mass [12,13]. To differentiate loculated pericardial effusion from a cardiac mass, CT or magnetic resonance imaging may be warranted [14]. In our case, confirmation of a loculated pericardial effusion was established due to the presence of contrast enhancement and the relationship with surrounding structures on CT imaging. Contrast-enhanced ultrasound may also be a good option to differentiate between the mass and loculated pericardial effusion [15]. As in the present case, color-Doppler is difficult to accurately evaluate the blood flow in a cardiac mas due to the artifact from the cardiac motion. Contrast-enhanced ultrasound enables sensitive and accurate blood flow assessment of cardiac masses and will be helpful in differentiating between cardiac tumours showing blood signals and loculated pericardial effusions without blood signals.

In conclusion, dogs with pericarditis may develop a loculated pericardial effusion with a severe mass effect, which may mimic a cardiac tumour. Loculated pericardial effusion is observed as a homogeneous structure without contrast enhancement on CT, and these features allow differentiation of loculated pericardial effusion from a cardiac mass.

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