Antemortem diagnosis of a left auricular appendage herniation through a partial pericardial defect in a dog with degenerative mitral valve disease

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Abstract

A 14-year-old neutered male crossbreed dog was presented for weakness, cough and weight loss. Cardiac auscultation revealed tachycardia, arrhythmia and a grade V/VI left apical systolic heart murmur. Thoracic radiographs showed a large homogeneous soft tissue opacity in close contact with the cardiac silhouette in the left cranioventral mediastinum. Cardiac evaluation showed atrial fibrillation, degenerative mitral valve disease and a dilated left auricular appendage outside the pericardium consistent with herniation through a partial pericardial defect. Seven months after diagnosis, an atrial septal defect secondary to acquired atrial septal rupture was identified. The dog was euthanized thirteen months after initial presentation because of unresponsive clinical signs of congestive heart failure.

Keywords: canine, heart, pericardium, ultrasound, X-ray

Antemortem Diagnose einer Herniation des linken Herzohrfortsatzes durch einen partiellen Herzbeuteldefekt bei einem Hund mit degenerativer Mitralklappenerkrankung


Schlüsselwörter: Herz, Herzbeutel, Hund, Röntgenstrahlen, Ultraschall
Case history

A 14-year-old 31.8 kg neutered male crossbreed dog was referred for a 10-day history of weakness, cough, and weight loss. The owner had adopted the dog when it was 4 years old, and the medical history prior to adoption was unknown. Physical examination showed mild tachypnea (40 breath/min), tachycardia (heart rate = 150 beats/min) with an irregular heart rhythm, and a grade V/VI left apical systolic heart murmur with an irregular normokinetic femoral arterial pulse. Respiratory auscultation revealed increased bronchovesicular lung sounds without identifiable crackles. Blood biochemistry analyses, including electrolytes, blood urea nitrogen and creatinine, were within normal limits. Based on the physical examination, an acquired cardiac disease, particularly degenerative mitral valve disease (DMVD), was suspected. First-line examinations, including thoracic radiographs and echocardiography, were therefore scheduled.

Radiography

Standard thoracic radiographs (Fig. 1A and 1B) showed the presence of a large homogeneous soft tissue opacity with convex borders in close contact with the cardiac silhouette in the left cranioventral mediastinum. A contralateral cardiac shift was observed with an upward rotation of the cardiac apex visible on the right lateral projection and the cardiac silhouette was subjectively increased in size. The trachea and carina were dorsally displaced. The cardiac apex was rounded which was unusual for a right lateral projection. Diameters of the pulmonary vessels were at the upper limits, but the lungs were clear. Chamber enlargement was difficult to assess due to the unusual rotation of the cardiac silhouette. All these findings were consistent with the presence of a left cranioventral mediastinal mass in close contact with a markedly enlarged cardiac silhouette, but without any obvious sign of congestive heart failure. Differentials for the cranioventral mediastinal mass included mediastinal neoplasm such as thymoma (given the ventral and caudal location), thymic or mediastinal lesion (e.g. cyst, abscess or hematoma), and, although less frequent, pericardial or cardiac lesions (e.g. pericardial cyst, pericardial or cardiac neoplasia, pericardial defect with cardiac aneurysm and herniation).

Electrocardiography

Standard 3-lead electrocardiography (ECG, Cardimax FX-7202, Fukuda Denshi, WA, USA) confirmed atrial fibrillation with tachycardia (ventricular rate = 160 beats/min).

Echocardiography

Conventional echocardiography and standard Doppler examination were performed on the awake dog, gently restrained in standing position, using continuous ECG monitoring with an ultrasound unit (Vivid 7 BT03, General Electric Medical System, Waukesha, Wis, USA) equipped with 3S (2.0–3.5 MHz) and 5S (2.8–5.0 MHz) phased-array transducers, as previously described (Chetboul et al., 2004). The bidimensional (2D) right parasternal 4-chamber view showed nodular thickening of the two mitral valve leaflets associated with thickened
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left chordae tendineae, consistent with DMVD lesions. Color Doppler mode examination confirmed severe mitral valve regurgitation on the left apical 4-chamber view, with a regurgitant fraction of 81% measured by proximal isovolumic surface area method (Gouni et al., 2007). A left ventricular dilation in diastole (66.2 mm) and systole (45.5 mm) (reference ranges RR: 41.5–45.1 mm and 25.8–28.4 mm, respectively, Gonçalvez et al., 2002) with normal fractional shortening (31%, RR: 30.1–49.0%, Chetboul et al., 2005) was observed on the left ventricular M-mode echocardiogram obtained from the right parasternal transventricular short-axis view. A left atrial (LA) enlargement was identified on the 2D right parasternal transaortic short-axis view (Fig. 2, LA on aorta ratio: 1.80 at end-diastole, RR: 0.52–1.13, Chetboul et al., 2005). The continuous-wave Doppler mode identified a high velocity mitral E wave (1.57 m/s, RR: 0.58–1.17 m/s, Chetboul et al., 2005), reflecting in part LA pressure overload. A high velocity systolic tricuspid regurgitation was also identified, allowing indirect estimation of systolic pulmonary arterial pressure (SPAP) using the simplified Bernoulli equation, as previously described (Serres et al., 2006). The SPAP was increased by up to 69 mmHg (RR: 15–25 mmHg, Kittleson and Kienle, 1998). Finally, the 2D right parasternal transaortic short-axis view showed a constriction of the basal portion of the left auricle (LAur) at the level of a partial pericardial defect (PD) associated with marked dilation of the LAur appendage (Fig. 2).

These findings were consistent with an advanced stage of DMVD associated with LAur appendage herniation (LAH) through a partial PD and atrial fibrillation. Due to the presence of PD, an abdominal ultrasound exam was performed to exclude other defects such as a diaphragmatic hernia, but it did not reveal any abnormalities.

**Therapy and follow-up**

The dog was medicated *per os* with furosemide (Dima­zon, Intervet, Beaucouze, France), 1 mg/kg BID, be­­nazepril (Fortekor, Novartis Santé Animale, Rueil Malmaison, France), 0.26 mg/kg SID, spironolactone (Prilactone, CEVA Santé Animale, Libourne, France), 0.52 mg/kg SID, and digoxin (Digoxine Nativelle, Valle Salimbene, Italia), 0.0015 mg/kg BID. A follow-up was scheduled every 2 months and included physical examination, echocardiography, ECG, thoracic radiographs and blood biochemistry. The dog was in good clinical condition and the treatment remained unchanged until seven months after the diagnosis, when the dog was presented with abdominal distention consistent with ascites secondary to pulmonary hypertension (SPAP=84 mmHg vs. 69 mmHg, RR: 15–25 mmHg, Kittleson and Kienle, 1998) and right-sided congestive heart failure. A 9.8-mm atrial septal defect (Fig. 3) consistent with an acquired atrial septal rupture was identified, leading to a left-to-right shunting between the two atrial cavities (peak systolic and diastolic velocities of the shunting flow: 2.2 m/s and 1.1 m/s, respectively). The LAH was of similar 2D echocardiographic aspect. The furosemide dosage was increased to 1.3 mg/kg TID and pimobendan (Cardisure, Eurovet, Bladel, Netherlands), 0.26 mg/kg BID was added to the current medical therapy. The dog was euthanized thirteen months after the initial diag­

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**Figure 2:** Bidimensional right parasternal transaortic short-axis view showing the left atrial (LA) and left auricular (LAur) dilation, with constriction of the basal portion of the LAur at the level of the pericardial defect (arrows) and LAur herniation. Ao, aorta; PT, pulmonary trunk; RVOT, right ventricular outflow tract.

**Figure 3:** Recording of the flow through the acquired atrial septal defect, using color-flow Doppler mode from the right parasternal long axis 4-chamber view. The shunting flow was continuously left-to-right (with peak velocities of 2.2 m/s in systole and 1.1 m/s in diastole). LA, left atrium; RA, right atrium.
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Discussion

The present report describes antemortem radiographic and echocardiographic features of a LAH through a partial PD in an adult dog with advanced DMVD and atrial fibrillation. Pericardial defects are defined as communications between the pericardial and pleural spaces (Gaag and Luer, 1977). These defects can be congenital or acquired (mainly traumatic), unique or multiple, small or large, and one side of the pericardium may even be totally absent (Gaag and Luer, 1977). Moreover, PD can be isolated or associated with other abnormalities such as abdominal wall, diaphragmatic or intra-cardiac defects, as previously described in Cocker Spaniel littermates (Bellah et al., 1989). A few cases of PD with or without heart incarceration have been previously described using radiography or necropsy (Kohler, 1958; Pallaske, 1959; Lehmann, 1960; Schieffer, 1962; Van den Ingh, 1977; Bohn, 1978; Milli and Unsuren, 1982; Brunnberg, 1989; Grabner, 1991). Additionally, a study published by Gaag and Luer in 1977 described necropsic findings in 8 dogs with isolated or multiple PD located on the left or the right side of the pericardium, and secondary cardiac constriction, especially when left or right ventricles were involved. Herniation of the right or left auricular appendage solely through a PD is a rare condition in dogs. Among the 8 PD described by Gaag and Luer (1977), only 2 dogs (one Dachshund and one crossbreed dog) had an auricular herniation, concerning the right and left auricle. In the dog with LAH, histological examination showed chronic auricular epicarditis. Twenty-eight years later, an auricular appendage herniation through a PD was diagnosed in 2 adult dogs (one Golden Retriever and one Lhassa Apso) by using radiography and computed tomographic angiocardiotics (Tomich et al., 2013). Although rare, LAH should therefore be included in the differential diagnosis of cranial mediastinal opacities detected by radiography, as previously suggested (Chapel et al., 2014). In the present report, transthoracic echocardiography allowed direct identification of the LAH by visualization of the extra-pericardial location of the LAur appendage through a large PD. Other imaging modalities that can facilitate detection of the origin of the mass include contrast-enhanced computed tomography (Tomich et al., 2013), which was not performed here.

In humans, permanent or intermittent herniations, strangulations or incarcerations of the heart, especially the ventricles, are associated with paroxysmal arrhythmias, ischemic incidents and hemodynamic alterations causing syncope, chest pain, and sudden death (Jones and McManus, 1984; Bennett, 2000). Therefore, if the PD circumscribes part of the ventricles, especially the body or apex, surgery is clearly warranted, even if the coronary circulation is not compromised (Bennett, 2000). Conversely, if the defect is confined to the upper heart border, strangulation or incarceration rarely occurs and the defect should be treated only in symptomatic patients (Bennett, 2000). In the present case, the clinical signs were attributable to DMVD rather than the LAH, and surgery was therefore not considered. Techniques for PD closure include primary closure or longitudinal pericardotomy for par-
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