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

C. Misbach[1], P. Pey[2], V. Gouni[1], E. Trehiou-Sechi[1], A. M. P. Petit[1], J.-L. Pouchelon[1, 3], V. Chetboul[1, 3]

[1] Unité de Cardiologie d'Alfort et [2] Service d'Imagerie Médicale, Ecole Nationale Vétérinaire d'Alfort, Centre Hospitalier Universitaire Vétérinaire d'Alfort, Maisons-Alfort, France, [3] INSERM, U955, Equipe 03, Créteil, France

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 Herzbeutel-defekt bei einem Hund mit degenerativer Mitralklappenerkrankung

Ein 14 jähriger kastrierter Mischling Rüde wurde wegen Schwäche, Husten und Gewichtsverlust vorgestellt. Bei der Herzauskultation fielen Tachykardie, Arrhythmie und ein apikales systolisches Herzgeräusch von Grad V/VI links auf. Röntgenbilder vom Thorax zeigten eine grosse homogene Weichteilverschattung in engem Kontakt mit der Herzsilhouette im linken kranioventralen Mediastinum. Eine kardiologische Untersuchung zeigte Vorhofflimmern, degenerative Mitralklappen-Endokardiose und ein dilatiertes linkes Herzohr ausserhalb des Perikardiums, was auf einen partiellen Perikardbruch hinweist. Sieben Monate nach der Diagnose, wurde ein atrialer Septumdefekt sekundär zu einer erworbenen Septumruptur identifiziert. Der Hund wurde dreizehn Monate nach der ersten Vorstellung wegen Verschlechterung der Herzinsuffizienz eingeschläfert.

Schlüsselwörter: Herz, Herzbeutel, Hund, Röntgenstrahlen, Ultraschall

DOI 10.17236/sat00063

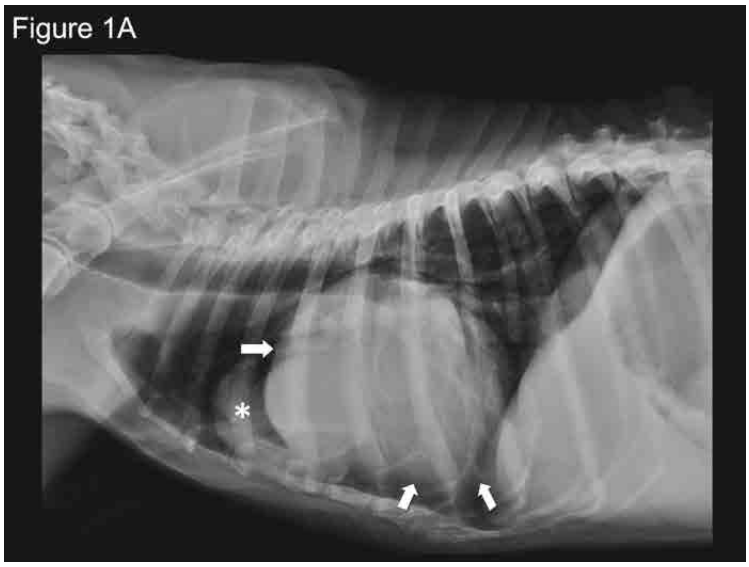
Received: 07.09.2015
Accepted: 19.01.2016

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

C. Misbach et al.

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.



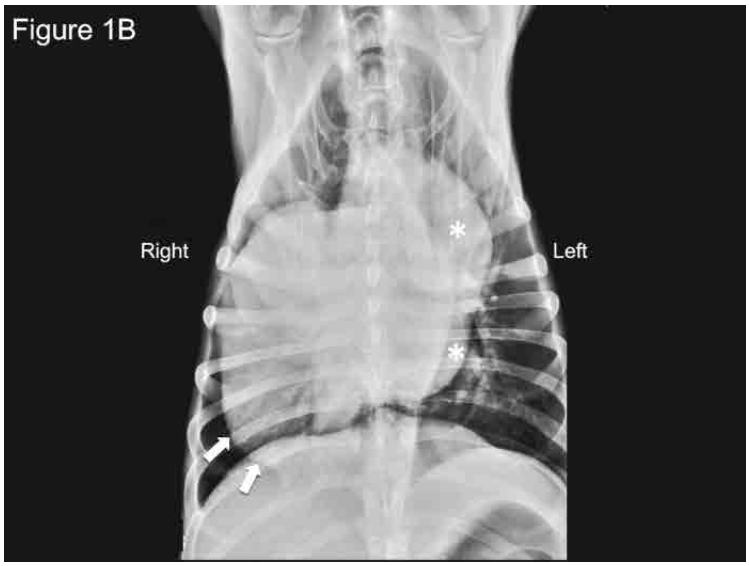


Figure 1 (1A and 1B): Right lateral (1A) and ventrodorsal (1B) radiographs of the thorax showing the presence of a voluminous soft tissue mass (asterisk) silhouetting with the enlarged cardiac shadow (arrows). The cardiac silhouette demonstrates an upward rotation on the lateral view and a contralateral shift on the ventrodorsal view.

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

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-

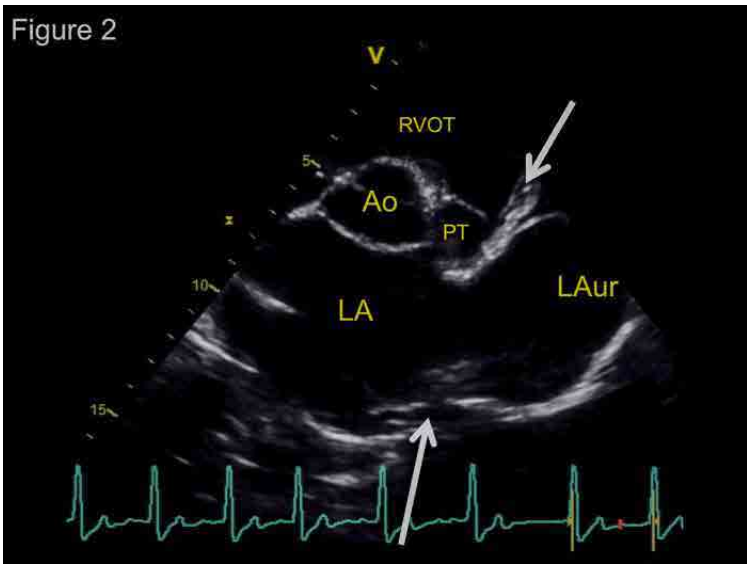


Figure 2: Bidimensional right parasternal transaortic short-axis view showing the left atrial (LA) and left auricular (LAur) dilation, with constriction of the basal portion of the LAur at the level of the pericardial defect (arrows) and LAur herniation. Ao, aorta; PT, pulmonary trunk; RVOT, right ventricular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

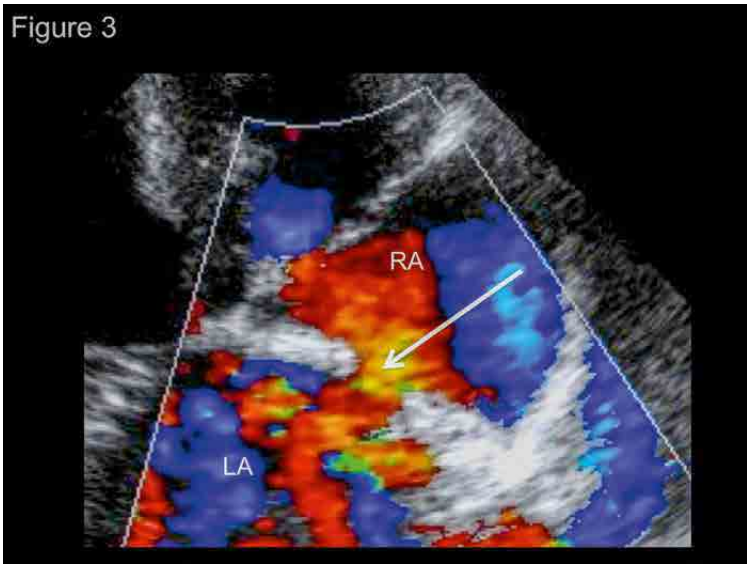


Figure 3: Recording of the flow through the acquired atrial septal defect, using color-flow Doppler mode from the right parasternal long axis 4-chamber view. The shunting flow was continuously left-to-right (with peak velocities of 2.2 m/s in systole and 1.1 m/s in diastole). LA, left atrium; RA, right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

nosis because of cachexia and unresponsive clinical signs of congestive heart failure. The owners declined necropsy.

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 angiocardiograms (Schwarz et al., 2005). A more recent report described LAH through a partial PD in a syncopal dog with DMVD and rapid runs of focal atrial tachycardia (Chapel et al., 2014).

In the present report, since the past history of the dog was unknown, the origin of the PD (congenital or acquired) could not be determined but herniation of the LAur was probably enhanced by the LA pressure overload secondary to severe mitral valve regurgitation. Interestingly, this rise in LA pressure, associated with jet lesions on the atrial wall, also led to an acquired atrial septal defect secondary to rupture of the atrial septum seven months after the initial diagnosis, as previously described in dogs with DMVD (Peddle and Buchanan, 2010). This LA pressure overload was confirmed by continuous Doppler mode evaluation of the shunting flow across the atrial septal defect. This showed a continuous left-to-right flow with a peak systolic velocity of 2.2 m/s, reflecting a high systolic pressure gradient between the two atrial chambers (19 mmHg, normal maximal value of 10 mmHg, Boon, 2011). As reported in humans (Misthos et al., 2009) and in an 8-year-old Golden retriever (Schwarz et al., 2005), auricular herniation can be associated with supraventricular arrhythmias including atrial fibrillation, independent of the LA size. Nevertheless, the marked LA dilation secondary to mitral valve regurgitation may also have contributed to such tachyarrhythmia.

In the present case, the radiographic diagnosis of LAH was challenging given the left location of the cranioventral mediastinal mass, which was unusual for a PD and also given the size of the opacity (i.e. due to abnormal size of the LAur owing to severe DMVD). The only specific features, based on human literature, that would have suggested PD as first probable hypothesis, would have been the unusual position of the heart within the thorax with cardiac rotation and chamber displacement. The convex left heart border with left hilar prominence and a border-forming cranially displaced LAur is actually a known feature of partial left PD with LAH in human imaging (Tucker et al., 1963; 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 syncopal episodes, dyspnea, 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-

tial PD, or pericardioplasty for absent pericardium (Van Son et al., 1993; Gatzoulis et al., 2000). Additionally, some authors advocate partial PD enlargement to relieve tension on the pericardial rim and prevent restrictive herniation (Kaneko et al., 1998; Drury et al., 2007), with special care regarding the phrenic nerve, especially when patch sutures are considered (Kaneko et al., 1998).

A limitation of this case is that no necropsy and histopathology examinations were undertaken although they would have been informative, especially to detect possible microscopic lesions of the LAur appendage attributable to the LAH (e.g., ischemic, necrotic or inflammatory). However, this report provides an original description of antemortem LAH through a PD, which remains a rare condition in small animals.

Acknowledgments

The authors would sincerely like to thank Dr Annalisa Giovannini and Dr Mathieu Raillard (DKV Anästhesie Abteilung - Vetsuisse Fakultät - Universität Bern) for the English to German translation.

References

Bellah J. R., Spencer C. P., Brown D. J., Whitton D. L.: Congenital cranioventral abdominal wall, caudal sternal, diaphragmatic, pericardial, and intracardiac defects in Cocker Spaniel littermates. J. Am. Vet. Med. Assoc. 1989, 194: 174–176.

Bennett K. R.: Congenital foramen of the left pericardium. Ann. Thorac. Surg. 2000, 70: 993–998.

Bohn F.: Clinically diagnosed rupture of the pericardium in a dog. Prakt. Tierarzt. 1978, 59: 513–516.

Boon J. A.: Congenital Shunts and AV Valve Dysplasia. In: Manual of Veterinary Echocardiography. 2nd ed. Eds. Wiley-Blackwell, Baltimore, USA, 2011: 437–475.

Brunnberg L.: Pericardial rupture with partial luxation of the heart in a dog. Case report. Kleintierprax. 1989, 34: 271–274.

Chapel E., Russel D., Schober K.: Partial pericardial defect with left auricular herniation in a dog with syncope. J. Vet. Cardiol. 2014, 16: 133–139.

Chetboul V., Athanassiadis N., Concordet D., Nicolle A., Tessier D., Castaignet M., Pouchelon J. L., Lefebvre H. P.: Observer-dependent variability of quantitative clinical endpoints: the example of canine echocardiography. J. Vet. Pharmacol. Ther. 2004, 27: 49–56.

Chetboul V., Sampedrano Carlos C., Concordet D., Tissier R., Lamour T., Ginesta J., Gouni V., Nicolle A. P., Pouchelon J. L., Lefebvre H. P.: Use of quantitative two-dimensional color tissue Doppler imaging for assessment of left ventricular radial and longitudinal myocardial velocities in dogs. Am. J. Vet. Res. 2005, 66: 953–961.

Drury N. E., De Silva R. J., Hall R. M., Large S. R.: Congenital defects of the pericardium. Ann. Thorac. Surg. 2007, 83: 1552–1553.

Gaag I. V. and Luer J. T.: Eight cases of pericardial defects in the dog. Vet. Pathol. 1977, 14: 14–18.

Gatzoulis M. A., Munk M. D., Merchant N., Van Arsdell G. S., McCrindle B. W., Webb G. D.: Isolated congenital absence of the pericardium: clinical presentation, diagnosis and management. Ann. Thorac. Surg. 2000, 69: 1209–1215.

Gonçalves A. C., Orton E. C., Boon J. A., Salman M. D.: Linear, logarithmic, and polynomial models of M-mode echocardiographic measurements in dogs. Am. J. Vet. Res. 2002, 63: 994–999.

Gouni V., Serres F. J., Pouchelon J. L., Tissier R., Lefebvre H. P., Nicolle A. P., Sampedrano Carlos C., Chetboul V.: Quantification of mitral valve regurgitation in dogs with degenerative mitral valve disease by use of the proximal isovelocity surface area method. J. Am. Vet. Med. Assoc. 2007, 231: 399–406.

Grabner A.: Ventricular tachycardia due to rupture of the pericardium in a dog. Kleintierprax. 1991, 36: 295–302.

Jones J. W. and McManus B. M.: Fatal cardiac strangulation by congenital partial pericardial defect. Am. Heart. J. 1984, 107: 183–185.

Kaneko Y., Okabe H., Nagata N.: Complete left pericardial defect with dual passage of the phrenic nerve: a challenge to the widely accepted embryogenic theory. Pediatr. Cardiol. 1998, 19: 414–417.

Kittleson M. D. and Kienle R. D.: Pulmonary arterial and systemic arterial hypertension. In: Small animal cardiovascular medicine. Eds St Louis, Mosby, 1998, 433–449.

Kohler H.: Ruptur des Herzbeutels be im Hund. Dtsch. Tierärztl. Wochenschr. 1958, 65: 489.

Lehmann H.: Über einen Fall von Strangulation der Herzspitze beim Hund. Prakt. Tierärzt. 1960, 10: 434.

Milli U. H. and Unsuren H.: Incarceration of the heart as a result of pericardial rupture in a dog. Veteriner. Fakultesi. Dergisi. 1982, 29: 214–218.

Misthos P., Neofotistos K., Drosos P., Kokotsakis J., Lioulias A.: Paroxysmal atrial fibrillation due to left atrial appendage herniation and review of the literature. Int. J. Cardiol. 2009, 133: 122–124.

Pallaske G.: Traumatische Perforation des Herzbeutels beim Hund. Dtsch. Tierärztl. Wochenschr. 1959, 66: 303.

Peddle G. D. and Buchanan J. W.: Acquired atrial septal defects secondary to rupture of the atrial septum in dogs with degenerative mitral valve disease. J. Vet. Cardiol. 2010, 12: 129–134.

Schieffer B.: Ruptur des Herzbeutels mit nachfolgender Inkarzeration des Herzens. Kleintierprax. 1962, 7: 101.

Schwarz T., Willis R., Summerfield N. J., Doust R.: Aneurysmal dilatation of the right auricle in two dogs. J. Am. Vet. Med. Assoc. 2005, 226: 1512–1515.

Serres F. J., Chetboul V., Tissier R., Carlos Sampedrano C., Gouni V., Nicolle A. P., Pouchelon J. L.: Doppler echocardiography-derived evidence of pulmonary arterial hypertension in dogs with degenerative mitral valve disease: 86 cases (2001–2005). J. Am. Vet. Med. Assoc. 2006, 229: 1772–1778.

Tomich J., Bhasin M., Philpott J.: The role of cardiac magnetic resonance imaging in the evaluation of congenital pericardial defects. Ann. Thorac. Surg. 2013, 95: 2178–2180.

Tucker D. H., Miller D. E., Jacoby W. J. Jr.: Congenital partial absence of the pericardium with herniation of the left atrial appendage. Am. J. Med. 1963, 35: 560–565.

Van den Ingh T. S.: Pericardial defect with incarceration of the heart in a dog. Schweiz. Arch. Tierheilk. 1977, 119: 473–476.

Van Son J. A., Danielson G. K., Schaff H. V., Mullany C. J., Julsrud P. R., Breen J. F.: Congenital partial and complete absence of the pericardium. Mayo. Clin. Proc. 1993, 68: 743–747.

Corresponding author

C. Misbach
DVM, PhD. Université Paris-Est
Ecole Nationale Vétérinaire d'Alfort
Unité de Cardiologie d'Alfort
Centre Hospitalier Universitaire Vétérinaire d'Alfort
7 avenue du général de Gaulle
94704 Maisons-Alfort cedex, France
Phone: +33 6 70 94 16 23.
E-Mail: charlotte.misbach@gmail.com