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Zi Ping Leong

Yoshiaki Hikasa

Case Report

Double-chambered left ventricle in a symptomatic cat

Zi Ping Leong[1] Yoshiaki Hikasa[1,2*]

Abstract

This case report describes a rare presentation of a cardiac anomaly in an 8-year-old, spayed female domestic short-haired cat presented with heart failure. Complete diagnostic work-ups which included chest radiography, electrocardiogram, echocardiography, and serum cardiac biomarker levels were performed. On echocardiography, a band of tissue was found extending in the left ventricular chamber, dividing the chamber into basal and apical portions, giving rise of a diagnosis of progressive double-chambered left ventricle. Suggestion to treat tachycardia and pulmonary edema was given to the veterinarian who referred the case. However, the cat was found dead one month later.

Keywords: congenital heart disease, congestive heart disease, double-chambered left ventricle, moderator band

[1]*The United Graduate School of Veterinary Science Yamaguchi University, 1677-1, Yoshida, Yamaguchi 753-8515, Japan*
[2]*Laboratory of Veterinary Internal Medicine, Joint Department of Veterinary Medicine, Faculty of Agriculture, Tottori University, 4-101 Koyama Minami, Tottori 680-8553, Japan*
***Correspondence**: hikasa@muses.tottori-u.ac.jp*

Introduction

Double-chambered left ventricle (DCLV) is an extremely rare congenital cardiac disorder characterized by an anomalous muscular bridge which separates the left ventricular (LV) cavity into two subunits, whereas double-chambered right ventricle (DCRV) has an intra-cavitary obstruction in the right ventricle and is often progressive (Pongiglione et al., 1982). Many human patients with DCLV are asymptomatic, and the disease is non-progressive (Nacif et al., 2016). Given the rarity of this anomaly, DCLV has unknown pathogenesis, although hypoplasia of the regional myocardial intra-trabecular sinusoids has been suggested (Gerlis et al., 1981). To date, only a few cases in human literature (Gerlis et al., 1981; Sanz et al., 2004; Breithardt et al., 2008; Makkuni et al., 2010; Nacif et al., 2010; Zhang et al., 2012; Bejiqi et al., 2013) and one case of a Siamese cat in veterinary literature (Smith et al., 2004) have been reported.

Case description

An 8-year-old, spayed female domestic short-haired cat was referred to the hospital for a diagnosis of an undetermined heart disease. In March 2015, when a grade II murmur was first auscultated as an incidental finding by a private veterinarian, the cat did not exhibit any clinical signs of heart disease. Neither hematology nor serum biochemistry revealed any remarkable findings to suggest a likely diagnosis (Table 1). The cat had negative antibody titers for feline leukemia (FeLV) and immunodeficiency (FIV) viral infections. However, serum N-terminal pro-brain natriuretic peptide (NT-proBNP) levels in January 2016 increased by almost 6-folds compared with those in April 2015 (Table 1). Since January 2016, the cat had been treated with enalapril (Enacard® 1 mg, Merial, United Kingdom), 0.26 mg/kg, every 12 hours, PO.

Table 1 Haematological, serum biochemical, and cardiac biomarker parameters

Parameters	Reference Range	13/04/2015	27/08/2015	29/01/2016	28/02/2016
Red blood cell (10^6/mm^3)	5-10	10.5	10.4	11.6	11.0
Packed cell volume (%)	30-45	43	40	43	43
White blood cell (/mm^3)	5500-19500	7500	9200	8000	7800
Neutrophil (/mm^3)	2500-12500	4350	4602	4600	4334
Lymphocyte (/mm^3)	1500-7000	2550	3036	2720	2686
Monocyte (/mm^3)	0-850	225	480	120	158
Eosinophil (/mm^3)	0-1500	375	1012	560	672
Platelet (10^3/mm^3)	300-800	203	232	159	170
Plasma protein (g/dL)	5.7-7.8	7.3	-	-	-
Albumin (g/dL)	2.3-3.6	3.0	-	-	-
Alanine aminotransferase (U/L)	22-84	52	65	66	56
Aspartate aminotransferase (U/L)	13-51	30	-	-	-
Alkaline phosphatase (U/L)	38-185	96	80	63	63
Cholesterol (mg/dL)	89-176	333	318	233	245
Triglyceride (mg/dL)	17-104	45	40	63	20
Glucose (mg/dL)	71-148	127	110	125	114
Blood urea nitrogen (mg/dL)	17.6-32.8	25.6	23.2	29.5	20.4
Creatinine (mg/dL)	0.8-1.8	1.0	1.6	1.4	1.4
Serum N-terminal pro-brain natriuretic peptide (ρmol/L)	0-100	47	110	278	-

The cat was bright and alert with a good body condition score (BCS 3/5), but showed tachypnea (78 breaths/min). Chest auscultation revealed tachycardia (heart rate 250 beats/min), a grade IV/VI continuous systolic heart murmur in the left thoracic region and harsh lung sounds. Blood pressures at systole and diastole were 133.2 mmHg and 90.6 mmHg, respectively. Electrocardiogram revealed supraventricular tachycardia and tall R waves (1.26 mV, RR < 0.9 mV) for lead II. On the right lateral view of the thoracic radiograph (Fig. 1), the enlarged cardiac silhouette appeared as a valentine shape with a vertebral heart score of 9 (VHS; RR < 8.0). The left atrium was noticeably enlarged, and the lung fields showed a vascular pattern consistent of pulmonary edema. From the ventro-dorsal view (Fig. 2), a right deviation of the heart and vasculo-interstitial pattern of the left caudal lung fields were also prominent.

Echocardiographic examination revealed that the left atrium was severely dilated with a dimension of 22.8 mm dorsoventrally and 35.8 mm horizontally and a left atrial to aortic root ratio (LA: Ao) of 3.59 (RR 0.88-1.70). Within the left ventricular (LV) chamber, a band of tissue measuring 3.2 mm dorsally and 4.8 mm ventrally in thickness was found extending from the left heart apex to the interventricular septum distal to the septal mitral annulus, which partitioned the LV chamber into basal and apical portions (Fig. 3). Both the basal and apical sub-chambers were next to each other. Both chambers contracted and dilated simultaneously. On color Doppler echocardiography (Fig. 4), the basal sub-chamber was observed to be connected to the LA and the Ao, and communicated through a central fenestration of the shelf with the apical sub-chamber, having no direct communication with the LA or the Ao. Turbulent jets flowing between these two sub-chambers recorded a peak systolic velocity of 4.69 m/s and a peak diastolic velocity of 2.32 m/s, which gave rise to a peak systolic pressure gradient of 87.98 mmHg from the apical portion to the basal portion of the tissue shelf. Furthermore, mitral valve regurgitations (5.27 m/s) and a high transmitral

inflow (E wave: 1.63 m/s, indicating a high LA pressure) were also detected. Based on the above findings, a diagnosis of DCLV with concurrent mitral valve insufficiency and congestive left heart failure was made. Recommendations were given to the private veterinarian to initiate medications such as beta-blockers and diuretic agents to control tachycardia and pulmonary edema, respectively. One month later, the cat was found dead in the owner's garden.

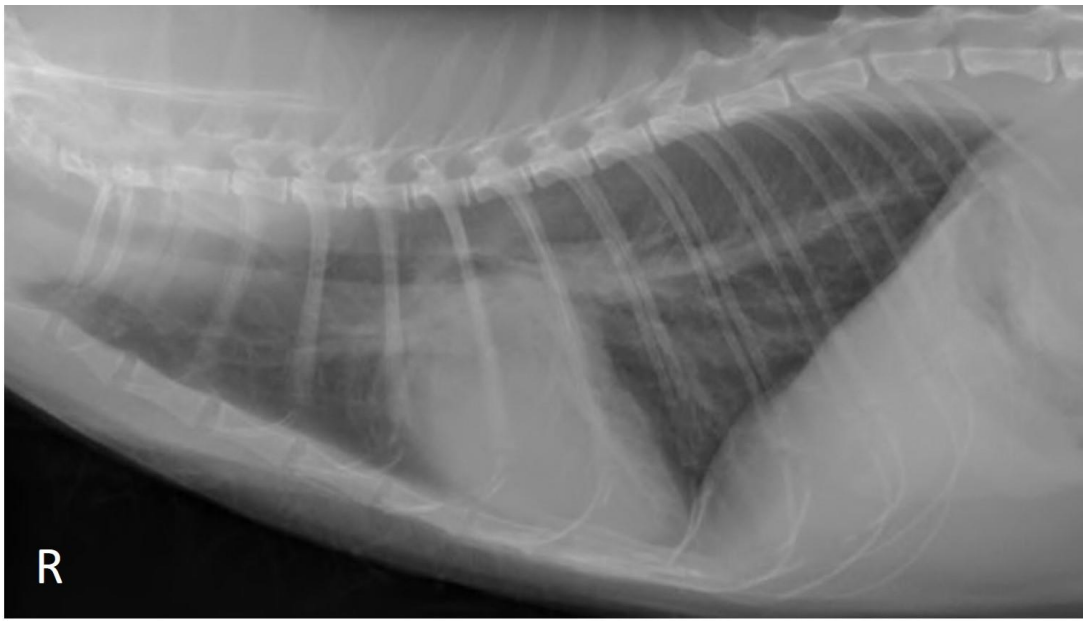

Figure 1 Right lateral view shows a globoid heart with a markedly enlarged left atrium and vascular lung pattern.

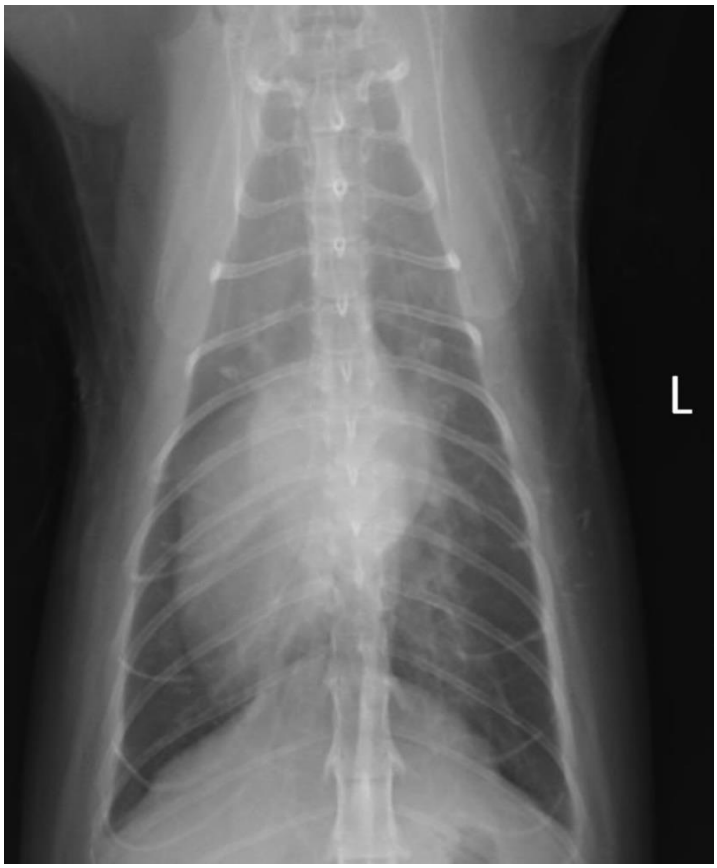

Figure 2 Ventro-dorsal view reveals a right deviation of the heart, a markedly enlarged left atrium and vasculo-interstitial lung pattern.

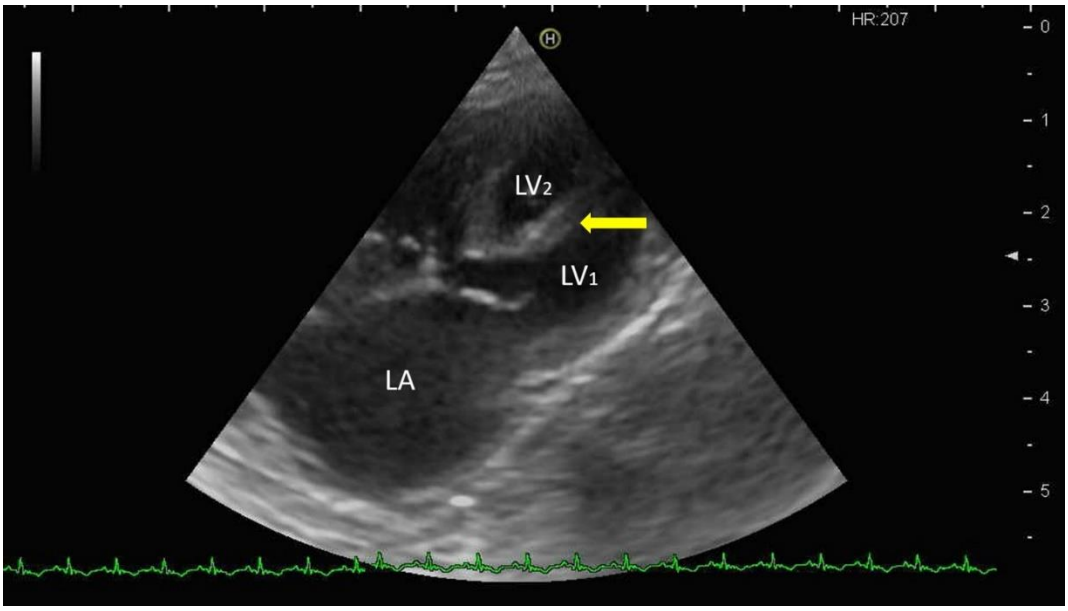

Figure 3 Modified right parasternal long axis view. Note the muscular band within the left ventricle (LV) which separates the LV chamber into apical and basal portions (arrow). The left atrium (LA) was markedly enlarged. LA = left atrium; LV_1 = basal sub left ventricular chamber; LV_2 = apical sub left ventricular chamber.

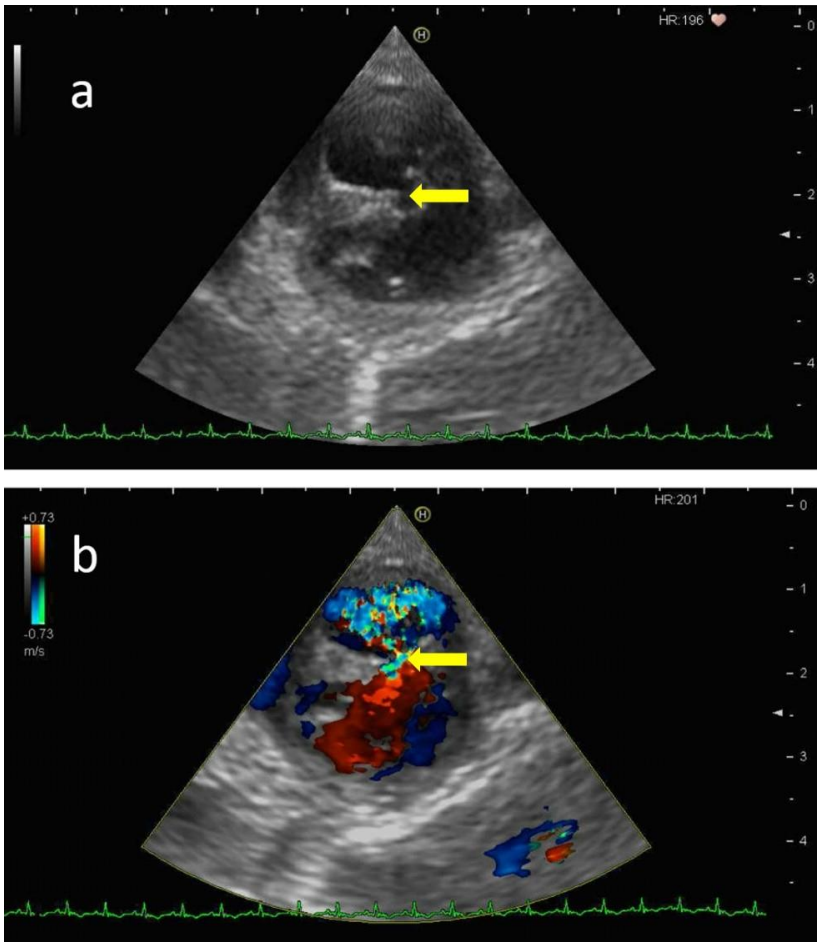

Figure 4 Right parasternal short axis view clearly shows the sub-division of the left ventricular chamber by a thick isoechoic shelf (a). On the color Doppler, the blood flowed through the wide central fenestration (arrow) across the shelf (b).

Discussion

To the best of our knowledge, this is the first case in veterinary literature to report a DCLV case in a cat showing progressive congestive heart failure (CHF). In year 2004, Smith and colleagues (2004) reported DCLV in a Siamese cat. The cat was asymptomatic and did not exhibit any signs of CHF. Its apical and basal sub-chambers were neither in series nor in parallel, with a peak systolic pressure gradient of 44 mmHg. In contrast, the cat presented in our case exhibited tachypnea and left CHF of approximately a five-fold increase in the serum NT-proBNP levels within 1 year and 3 months. The sub-chambers were

parallel to each other, recording a high peak systolic pressure gradient of 100 mmHg. Nevertheless, several findings were also observed similar to that by Smith and colleagues (2004), for example, both cats had tachycardia, left atrial enlargement and did not show any conduction disturbance on the electrocardiogram, except for a high R-wave amplitude in lead II. We speculate that the high R wave might be attributable to the presence of the anomalous muscular tissue in the LV, which further increased the depolarization impulse.

Two important differential diagnoses of DCLV include LV aneurysm and LV diverticulum. The LV aneurysm is a discrete region of the ventricular wall that is thinner and weaker than the adjacent myocardial structures, where it bulges outward and exhibits either akinesis or dyskinesis. On the other hand, the LV diverticulum has an outpouching structure that contains the endocardium, myocardium and pericardium and displays normal contraction (Makkuni et al., 2010). In the present case, the two sub-chambers were formed by the separation of a thick muscular band. The LV had a central opening to permit intra-cavitary communication. The sub-chambers showed synchronous contractile motion and did not extend beyond the left ventricle. These findings are consistent with human cases of DCLV, suggesting a diagnosis of DCLV, rather than LV aneurysm or diverticulum. In human cases of DCLV, the separated chambers are in parallel with no significant pressure gradient as both chambers contract synchronously. While the cat described in this case had parallel LV sub-chambers and a high intra-cavitary systolic pressure gradient on the opposite side, Bejiqi and colleagues (2013) also detected a high systolic gradient between the two left chambers (67.6 mmHg) in a human with DCLV concurrent with idiopathic hypertrophic cardiomyopathy. The cause of CHF remains unknown because of the non-progressive nature of DCLV as reported by Nacif and colleagues (2010). We hypothesized that the cat might be suffering from concurrent cardiomyopathy, of which the potential role of the muscular band in the pathogenesis of this condition remains unknown.

Other differential diagnosis was the endomyocardial form of restrictive cardiomyopathy (RCM) which is hallmarked by a severe endomyocardial scar that may diffusely involve the LV or appear as a distinct lesion bridging the ventricular septum and LV free wall. On echocardiography, a dense, bright endocardial scar, normal or reduced LV cavity dimensions or severe endocardial fibrosis that obliterates the apical LV cavity are often observed (Fox, 2004). Although we were unable to confidently rule out the endomyocardial form of RCM from DCLV antemortem, the LV band in this cat did not resemble the scar tissue observed via echocardiography. Therefore, a DCLV case with concomitant RCM as a diagnosis was unlikely. Nevertheless, a pathological examination is necessary to confirm the diagnosis, although it was not carried out in this case.

The anomalous shelf in the cat might be the result of the fusion of excessive LV bands, as suggested by Smith and colleagues (2004). More commonly found on the right side of the heart (Boon, 2011), the false tendon(s) or the "moderator band" crosses the ventricular cavity and connects the septum, few walls or papillary muscles. However, not all false tendons create problems, and they are considered to be a normal anatomical feature in humans as well as six animal species (Gerlis et al., 1981). Due to scarcity of this cardiac anomaly, the expected survival time in cats with DCLV has not been documented in veterinary literature. However, clinical deterioration, manifestation of CHF and suspected concurrent cardiomyopathy pointed towards a guarded prognosis of survival for the cat.

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บทคัดย่อ

Double-chambered left ventricle ในแมวที่มีภาวะหัวใจล้มเหลว

ซี ปิง ลอง[1] โยชิอากิ ฮิกาซา[1,2]

รายงานสัตว์ป่วยนี้นำเสนอความผิดปกติของหัวใจที่พบยาก เคสนี้เป็นแมวที่ทำหมันแล้วอายุ 8 ปี พันธุ์ domestic short-haired และมีอาการหัวใจล้มเหลว การวินิจฉัยอย่างละเอียดรวมถึงการตรวจ ภาพรังสีช่องอก คลื่นไฟฟ้าหัวใจ ภาพคลื่นเสียงสะท้อนความถี่สูง และดัชนีชี้วัดของหัวใจในซีรัม ภาพคลื่นเสียงสะท้อนความถี่สูงพบแถบของเนื้อเยื่อแบ่งหัวใจห้องล่างซ้ายออกเป็นส่วน base และส่วน apex นำไปสู่การวินิจฉัยว่าเป็น double-chambered left ventricle สัตวแพทย์ให้คำแนะนำในการรักษาภาวะหัวใจเต้นเร็วกว่าปกติและภาวะน้ำท่วมปอด และส่งไปให้สัตวแพทย์ที่นำส่งเคสมา อย่างไรก็ตามแมวตัวนี้ได้เสียชีวิตลงหนึ่งเดือนต่อมา

คำสำคัญ: โรคหัวใจที่เป็นตั้งแต่เกิด ภาวะหัวใจล้มเหลว double-chambered left ventricle moderator band

[1]*The United Graduate School of Veterinary Science Yamaguchi University, 1677-1, Yoshida, Yamaguchi 753-8515, Japan*

[2]*Laboratory of Veterinary Internal Medicine, Joint Department of Veterinary Medicine, Faculty of Agriculture, Tottori University, 4-101 Koyama Minami, Tottori 680-8553, Japan*

***ผู้รับผิดชอบบทความ** E-mail: hikasa@muses.tottori-u.ac.jp*