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Fossa ovalis tear causing right to left shunting in a Cavalier King Charles Spaniel☆

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KEYWORDS
Atrial septal defect;
Fossa ovalis;
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Abstract Left atrial tear is an infrequent sequela of severe mitral regurgitation due to myxomatous mitral valve degeneration. Interatrial septal tear due to mitral regurgitation causing a left-to-right shunt is uncommon. Right to left shunting secondary to acute interatrial septal tear is very rarely reported in the human literature, and has not been reported in the veterinary literature in a dog. This case describes the clinical, radiographic, echocardiographic, gross pathologic, and histopathologic features of a dog presented in acute respiratory distress secondary to acute onset right to left shunting through the interatrial septum. This was later documented to be due to a tear in the septum secondary to tricuspid regurgitation and pulmonary hypertension. The presence of an acquired right to left shunting atrial septal defect is of clinical and prognostic significance, and should be considered in cases of acute respiratory distress.

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Abbreviations

MMVD myxomatous mitral valve degeneration

A 13 year-old spayed female Cavalier King Charles Spaniel was referred to the William R. Pritchard Veterinary Medical Teaching Hospital (VMTH) at the University of California, Davis for further evaluation of acute onset respiratory distress. The dog initially presented to a local emergency veterinary clinic with a 7-day history of excessive panting, and 2-h history of dyspnea. Pertinent medical history included a previous echocardiographic diagnosis of myxomatous mitral and tricuspid valve degeneration, moderate mitral regurgitation (i.e., moderate left atrial enlargement), and mild pulmonary hypertension (tricuspid regurgitation Vmax 3.5 m/s, PG 49 mmHg) 2 years prior to presentation. Prior echocardiographic examinations (starting from 4 years prior) were available for review and revealed no evidence of right atrial enlargement or right ventricular eccentric hypertrophy.

On presentation to the referral emergency clinic, a grade V/VI systolic heart murmur (not localized) was reportedly heard and soft respiratory crackles were reportedly ausculted in all lung fields. Thoracic radiography revealed severe cardiomegaly and moderate left atrial enlargement without evidence of pulmonary edema. Pulse oximetry was performed and revealed an SPO_2 on room air of 66% that increased to 88% in an oxygen cage (oxygen flow set to 10 L/min, FIO_2 not documented). A complete blood count revealed hematocrit 32.3%, reticulocytes 34.2 K/uL, white blood cells 19,590 K/uL, neutrophils 15,690 K/uL and the serum chemistry panel was unremarkable other than an elevated alanine transaminase (195 U/L, normal range 10—100). Despite the lack of radiographic evidence of pulmonary edema, left heart failure was diagnosed and the dog was treated overnight with a total of 12 mg/kg furosemide IV (3 $\times$ 4 mg/kg boluses over 10 h), 0.1 mg/kg butorphanol IV once, 0.27 mg/kg pimobendan PO once and supplemental oxygen. No improvement in respiratory rate or effort was noted over the hospitalization period and the dog was referred to the UC Davis VMTH Veterinary Cardiology Service the following morning for further evaluation.

On presentation, the dog was tachycardic (heart rate = 160 bpm), tachypneic (respiratory rate = 40—90 breaths per minute) with severely increased inspiratory effort, and normothermic (T = 101.3 F). The dog was overweight (9.4 kg with a body condition score of 7/9). Grade IV/VI left apical systolic and V/VI right apical systolic murmurs were ausculted, with occasional premature beats with pulse deficits. Femoral pulses were otherwise strong and synchronous. Fine crackles were ausculted in the right caudal lung field and a severely increased inspiratory respiratory effort, with increased abdominal effort, was noted. When placed in left lateral recumbency the dog became severely dyspneic and grossly cyanotic. A 6-lead electrocardiogram revealed tall P waves (0.5 mV) consistent with right atrial enlargement (p pulmonale), a normal P wave duration of 0.04 s, and a mildly prolonged PR interval (0.14 s). ST segment depression (0.4 mV) in lead II was also appreciated. The rhythm was a sinus tachycardia, with occasional single premature ventricular complexes of right ventricular origin (left bundle branch block morphology). Three-view thoracic radiography revealed severe cardiomegaly with moderate left atrial enlargement and no evidence of pulmonary venous distension or pulmonary edema. Due to respiratory distress, a limited echocardiographic examination was performed that revealed subjective evidence of severe right atrial enlargement and moderate right ventricular chamber enlargement. An irregular, mobile flap of tissue was evident in the region of the fossa ovalis (Fig. 1, Video 1). The main pulmonary artery and right pulmonary artery branch were moderately dilated (pulmonary artery:aorta ratio 1.4). A possible thrombus (hyperechoic lesion) was visualized in the distal portion of the right main branch of the pulmonary artery. Color flow Doppler interrogation

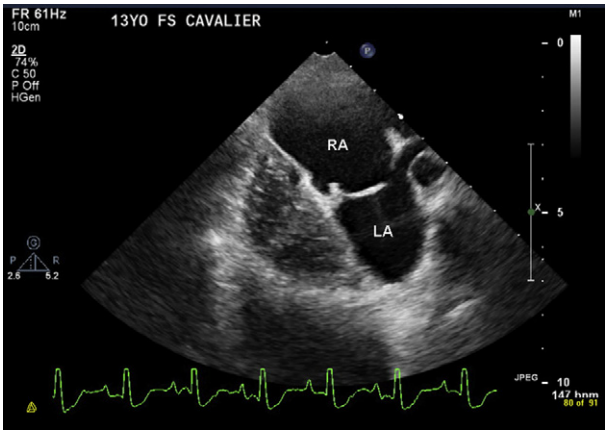

Figure 1 This is a limited right parasternal four-chamber view. Note the irregular flap of tissue in the region of the fossa ovalis.

revealed mild mitral insufficiency and moderate to severe tricuspid insufficiency. The jet of tricuspid insufficiency was directed toward the region of the fossa ovalis (Fig. 2). Continuous wave Doppler interrogation of the tricuspid insufficiency jet (performed in sternal recumbency due to respiratory distress) was consistent with moderate to severe pulmonary hypertension (peak velocity = 4.2 m/s; pressure gradient = 71 mmHg). Contrast echocardiography using 0.3% hydrogen peroxide solution (0.2 ml 3% hydrogen peroxide mixed with 2 ml heparinized saline) was performed, and confirmed the presence of a right-to-left shunt across the interatrial septum (Fig. 3). Given the poor prognosis, the owner elected euthanasia. A necropsy was performed.

On gross examination, the right atrium was severely enlarged and the mitral and tricuspid valve leaflets diffusely thickened, consistent with myxomatous degeneration. The left atrium was mildly to moderately enlarged. There was no evidence of chordae tendineae rupture. There were no grossly detectable thrombi present within the pulmonary arteries, however the lungs were diffusely firm. An approximately 0.5 cm × 0.75 cm full thickness tear was present in the region of the fossa ovalis (Fig. 4). The interatrial septal myocardium adjacent to the rupture was circumferentially thinned, measuring <0.1 cm thick and semi-transparent. The myocardium of the interatrial septum remote from the rupture was completely opaque.

Histologically, there was severe, segmental cardiomyocyte necrosis and myodegeneration along the edge of the rupture characterized by hypereosinophilia, loss of striations, myofiber fragmentation and vacuolization, prominent, centralized nuclei, and mineral deposition. Small to moderate amounts of hemorrhage dissected between cardiomyocytes. Additionally, abundant

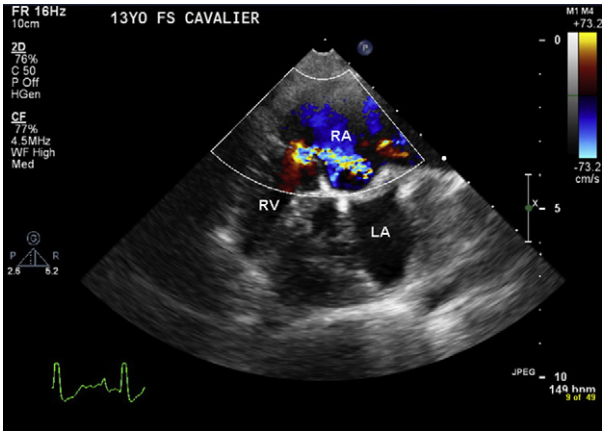

Figure 2 This is a right parasternal four-chamber view with color Doppler interrogation of the tricuspid valve. Note the jet of tricuspid insufficiency that is directed toward the defect in the atrial septum.

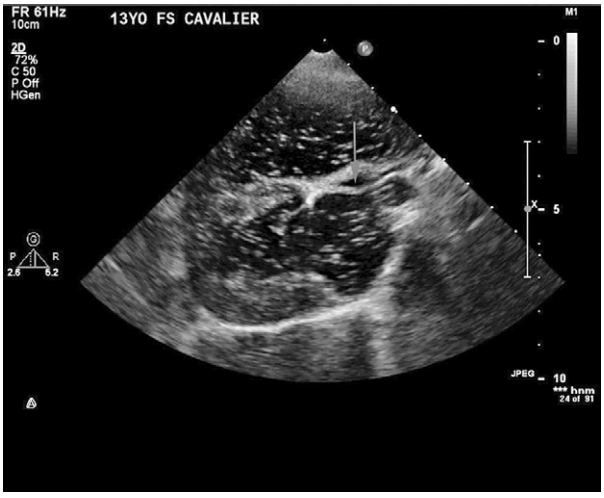

Figure 3 This is a bubble study from the right parasternal four-chamber view. Two ml of 0.3% hydrogen peroxide solution (0.2 ml 3% hydrogen peroxide mixed with 2 ml heparinized saline) was injected into the left cephalic vein. The bubbles are visualized crossing the atrial septum through the defect (marked by gray arrow).

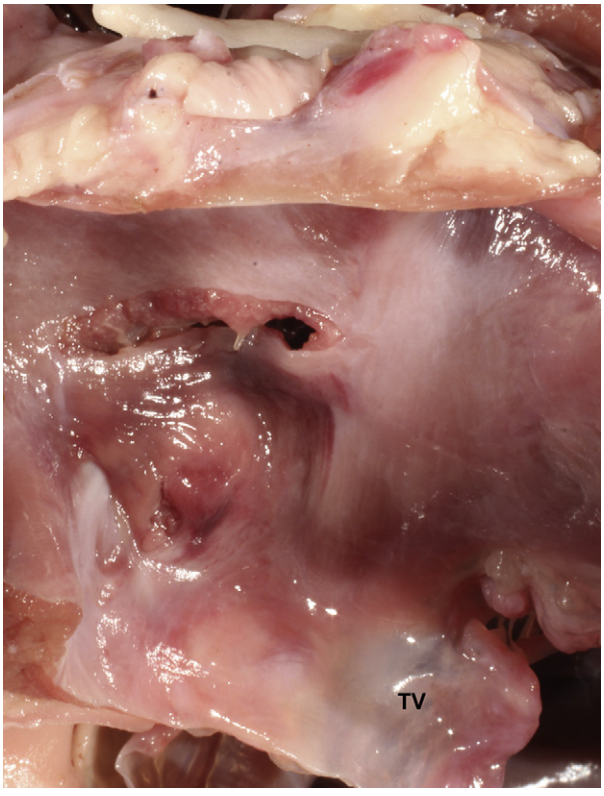

Figure 4 This is a dorsal view (from within the right atrium) of the atrial septum. A 0.5 cm × 0.75 cm tear is present in the region of the fossa ovalis.

fibrin and large numbers of neutrophils populated the myocardium adjacent to the rupture.

Mild to moderate, chronic, diffuse interstitial pulmonary fibrosis was present, with endothelial and tunica media hypertrophy and adventitial fibrosis of the pulmonary arterioles (Fig. 5) consistent with chronic pulmonary arterial hypertension.[1,2]

Discussion

This case characterizes a unique presentation of acute respiratory distress secondary to an acquired, right to left shunting atrial septal defect. In this dog, echocardiographic examination was consistent with a right to left shunt through a defect in the interatrial septum, and gross and histopathologic examination supported the diagnosis of an acute atrial tear. It is reasonable to expect that an acute tear was responsible for the sudden (i.e., within 2 h) decompensation and that hypoxemia due to the right-to-left shunt caused or exacerbated the dog's respiratory distress. This is especially likely given the presence of neutrophils and fibrin, both elements of acute inflammation. The cause of the 7-day history of panting is unknown. In the absence of evidence of congestive heart failure, or clinical history of respiratory disease, pulmonary thromboembolism would be a primary differential diagnosis but evidence to support this was not identified on necropsy.

Left atrial rupture secondary to severe myxomatous mitral valve degeneration (MMVD) is reported and is considered an uncommon cause of pericardial effusion, cardiac tamponade and sudden death in dogs.[2-7] Buchanan and Kelly[6] described left atrial splitting resulting from separation of endothelium from the subendothelial tissues and/or myocardium in a report of 22 dogs with mitral insufficiency. In this study, 7 of the dogs also had pericardial effusion. The location of the endothelial defects varied. However, the largest defects were most frequently noted in the caudal wall of the left atrium, a region where the regurgitant jet is commonly directed. An acquired atrial septal defect due to a tear in the interatrial septum was detected on necropsy in 3 dogs in the region of the fossa ovalis.[6] A similar lesion was described by Peddle and Buchanan[4] in a dog with an acquired atrial septal defect caudal to the fossa ovalis.

The etiology of left atrial rupture secondary to mitral insufficiency is unknown. Severe MMVD results in increased left atrial pressure and causes mechanical trauma from an eccentric jet of mitral insufficiency that may be a predisposing factor for spontaneous endocardial splitting.[4,6–8] The pathologic significance of endocardial jet lesions is uncertain, although jet lesions have been documented in dogs with left atrial rupture.[4] Although the fossa ovalis may represent a site of anatomical weakness that in theory may make it more likely to tear in the face of mechanical trauma and increased atrial pressure, most atrial tears occur in the lateral wall of the left atrium. The gross finding of a full thickness tear through the region of the fossa ovalis in the dog presented in the current report is similar to the dog described by Peddle et. al. in which that dog developed right heart failure secondary to left-to-right shunting through the interatrial defect. In our case, increased right atrial pressure secondary to tricuspid regurgitation and pulmonary hypertension, and mechanical trauma secondary to tricuspid regurgitation likely resulted in a tear of the fossa ovalis. The subsequent right to left shunting resulting in acute hypoxemia explained the dog's sudden onset of respiratory distress and cyanosis.

There are few reports in the human literature describing right to left shunting secondary to interatrial tear. Four reports[8–11] describe blunt chest trauma and secondary rupture of chordae tendineae of the tricuspid valve. None of the described patients had a history consistent with previous right to left shunting or known atrial septal defect. Necropsy was not performed in any of the patients, however it was suggested that the fossa ovalis is a site of anatomical weakness in the

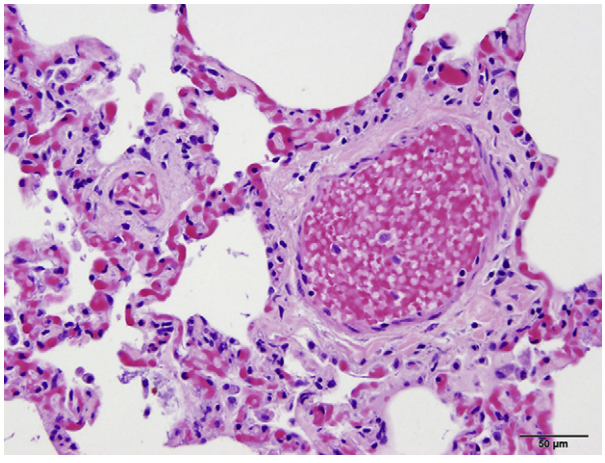

Figure 5 Pulmonary artery and arteriole are shown. Medial and adventitial hypertrophy of the pulmonary vasculature, typical of chronic pulmonary arterial hypertension is appreciated.

interatrial septum that may be prone to tear.[8] To the authors' knowledge, an acquired right to left shunting atrial septal tear secondary to tricuspid regurgitation and pulmonary hypertension in a dog has not been reported in the literature.

Conflict of interest

No conflict of interest.

Supplementary data

Supplementary data related to this article can be found online at http://dx.doi.org/10.1016/j.jvc.2012.07.004.

Table of video.

Video #	Brief title	Description
1	Irregular flap of tissue	This is a limited right parasternal four-chamber view. Note the irregular flap of tissue in the region of the fossa ovalis.

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