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# Right ventricular aneurysm and atrial septal defect in a cat

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## Abstract

This case report shows the development of a right ventricular aneurysm in a cat with a large atrial septal defect. Despite this complex cardiac pathology, the cat lived normally for more than 4 years and developed fatal congestive heart failure.

**Kew words:** Aneurysm - Cat - Atrial septal defect - Echocardiography.

A 14-month old Persian male cat with a history of chronic rhinitis was referred for an evaluation of acute intermittent hind leg lameness. On physical examination, the cat was thin (3.6 kg body weight), but normally developed. He was hyperthermic (40.2°C) and mildly tachypneic (60/min). The auscultated heart rate was 180/min, and a grade 3/6 systolic and soft grade 2/6 diastolic murmur was heard over the heart base on both sides of the thorax. The peripheral pulses were weak bilaterally, but there was no evidence of arterial thromboembolisation on clinical examination. Passive hip movements were painful without specific location. The blood pressure was normal[1] (118/68 mmHg, heart rate: 159 bpm). Severe cardiomegaly was evident on thoracic radiographs (Figures 1A and 1B). Pulmonary artery enlargement as well as dorsal displacement of the sternum (pectus excavatum) were also present. The electrocardiogram (ECG) had an abnormally widened QRS-complex (0.05 sec) with deep S-waves on leads I, II, III and aVF (Figure 1C), interpreted as atypical right ventricular hypertrophy pattern (RVH). Additional isolated premature contractions originating from the right ventricle were noticed. The standard echocardiographic examination[2] showed a dilatation of the right ventricle (RV) and atrium (RA). The thickness of the RV-free wall appeared to be normal. A large atrial septal defect (ASD) was seen and was illustrated by color flow Doppler as a low velocity (1.5 m/sec) left to right shunt across the defect (Figures 2A and 2B). On M-mode examinations, the left ventricular fractional shortening (FS: 37%) and left atrium to aortic root ratio were normal (1.12). Serological test for feline leukaemia virus (FeLV antigen), feline immunodeficiency virus (FIV-antibodies) and feline coronavirus (antibodies) were negative. CBC and serologic chemical parameters were normal.

The diagnosis of an ASD (probably associated with partial endocardial cushion defects) was made, complicated by an upper airway infection (no signs of bronchitis or peribronchitis on thoracic radiographs) and pulmonary overcirculation. The cat was treated with an ACE-inhibitor (benazepril 0.5 mg/kg SID, permanently)[3] and doxycycline 0.5 mg/kg BID for 10 days.

During the next 4 years, the right ventricular volume overload remained stable with the ACEI-therapy alone, even with many recurrences of chronic upper airway infections. The cat had 3 to 4 relapses of presumably feline upper respiratory tract infection per year despite adequate vaccinations. Each episode was treated successfully with enrofloxacin.

Forty-two months after the initial diagnosis of the cardiac malformation, the cat was represented with a mild hypothermia and dyspnea. He weighed 4.1 kg. Dullness of the percussion sounds were noticed over the right thoracic wall, as well as a cardiac arrhythmia. He had developed a unilateral, right-sided pleural effusion (Figure 3A), associated with permanent ventricular bigeminy on the ECG (Figure 3B). Cytological examination of the withdrawn liquid revealed a protein content of 20g/L and a specific gravity of 1.020, with non-degenerated neutrophiles, rare macrophages and monocytes, sometimes with erythrophagocytosis, and a few erythrocytes. This effusion was classified as a modified transudate, with mild hemorrhagic and non-septic inflammation, and was therefore compatible with congestive heart failure. The echocardiographic examination was quite similar to the previous one, but showed a more severe dilatation of the right ventricle and an additional aneurismal bulge of the upper parts of the right ventricular free wall, which probably had been present for a long time (Figure 2C). A diuretic (furosemide 0.5 mg/kg SID) was added to the treatment, and the cat developed mild prerenal azotemia (BUN 13.7 mmol/L, normal range: 3.6-10.7 mmol/L) without elevation

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of the plasma creatinine level. The remaining blood values were normal, inclusive a measurement of $T_4$.

Despite repeated thoracentheses (once a week during 2 months), it was not possible to prevent the thoracic effusion from reaccumulating, and the cat developed additional multifocal ventricular premature contractions (Figure 4). On repeated echocardiograms, neither signs of pulmonary hypertension nor signs of reversal of the shunt through the ASD were noticed. Six weeks after the first clinical signs of congestive heart failure, and 44 months after the initial diagnosis of ASD, the cat died during the transport to the clinic because of another episode of acute respiratory distress.

## Necropsy

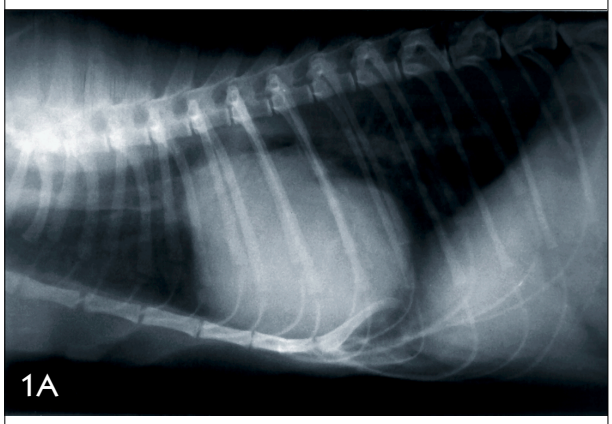
At post-mortem examination, there were subcutaneous edema, serosanguineous thoracic and abdominal effusions as well as pulmonary edema. The heart showed a severe eccentric hypertrophy of the right ventricle and atrium. An aneurysm with a base of 16 x 18 mm in diameter and 8 mm lateral extension was observed in the cranio-lateral free wall of the right ventricle (Figure 5). The ventricular wall was thinner in the aneurysm and measured about 1 mm of wall thickness (Figure 6A). The previously diagnosed ASD was confirmed and considered to be of a secundum type with approximately 8 mm in diameter. The annulus of the tricuspid valve was dilated. The left ventricle showed a slight concentric hypertrophy. Some fibrous adherences were observed between the pericardial sac and the right atrium. The liver was slightly enlarged with an irregular surface. Numerous cysts of about 0.5 to 7 mm in diameter were found in the cortex of both kidneys. Some cysts reached the medulla.

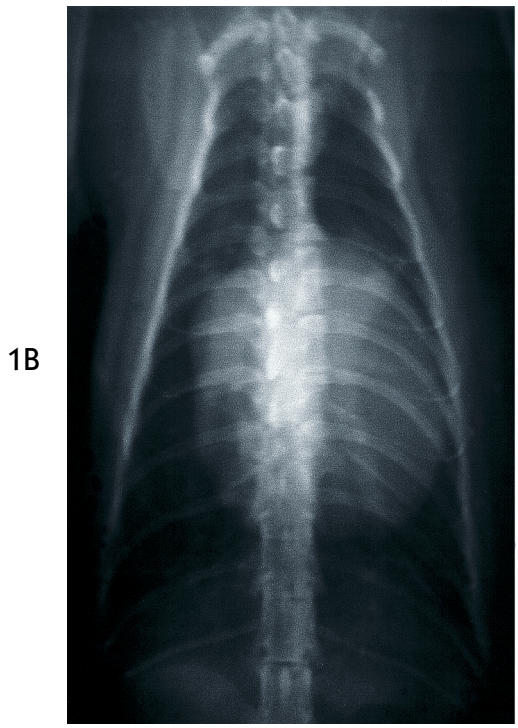
On histopathologic sections of the aneurysm, fewer than normal endocardial and myocardial cells were identified, and they had been partially replaced by fibroblastic cells and fibrocytes (Figures 6A and 6C). A slight round-cell myocarditis and a moderate amount of collagen deposition were also observed (Figures 6B and 6C). A horizontal section, above the aneurysm, did not reveal any modification of the epicardial vessels. The lung had slight amounts of interstitial fibrosis. The small pulmonary arteries were normal. The liver showed numerous foci of extramedullary haematopoiesis, hepatocellular fatty changes and a slight fibrosis of the centrolobular veins. In the kidneys the smaller cysts were delimited by a cubic epithelium, the bigger ones by a flattened epithelium. Some cysts contained an eosinophilic ground substance.

## Discussion

Right ventricular aneurysms are very rare in cats. Previous reports relate right ventricular aneurysm in cats with ARVC (arrhythmogenic right ventricular cardiomyopathy)[4] and left ventricular aneurysms with hypertrophic cardiomyopathy.[5] The development of a cardiac aneurysm is supposed to be secondary to ischemic cardiomyopathy. In humans, a true ventricular aneurysm is most often the consequence of a previous transmural myocardial infarction, or can also develop in patients who sustained a blunt chest injury.[6] It is defined as a segment of the ventricular wall that exhibits paradoxical systolic centrifugal expansion, has a broad neck, a wall thinner than the rest of the left ventricular wall, and is usually composed of fibrous tissue as well as necrotic muscular tissue, occasionally mixed with viable myocardium.[6,7] Pseudoaneurysms (false aneurysms) occur after localized myocardial rupture in which the hemorrhage is limited by pericardial adhesions and has a mouth that is

**Figure 1 - 1A, 1B.** Thoracic radiographs: bilateral cardiomegaly on the left lateral (1A) and dorsoventral (1B) views at day 0. Enlarged pulmonary arteries and dorsal displacement of the sternum (pectus excavatum) can bee seen. **1C.** Lead II Electrocardiogram at day 0 (25 mm/sec., 1 mV/10 mm) showing a widened QRS-complex with ventricular bigeminy.



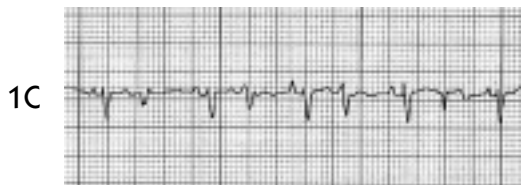
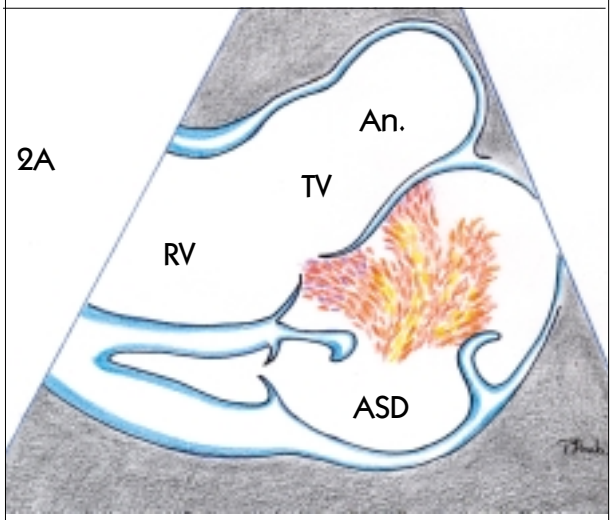
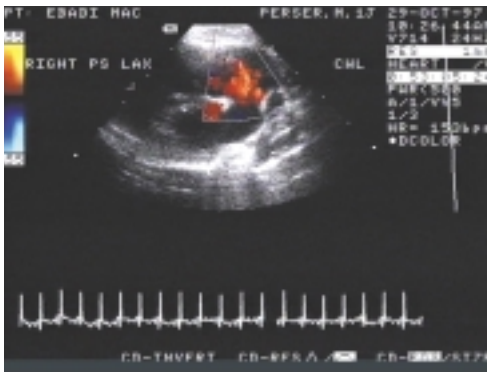



**Figure 2 -** Echocardiographic and Doppler examination.
**A.** Schematic drawing of the 2D right parasternal four-chamber view: eccentric hypertrophy of the right ventricle (RV) and atrium, aneurysm (An) in the free wall of the right ventricle and atrial septal defect (ASD). The left ventricle and left atrium are normal, and the tricuspid valve (TV) is displaced because of the aneurism.
**B.** Turbulent color Doppler flow illustrating the ASD (2D right parasternal four-chamber view).
**C.** Two dimensional echocardiogram 42 months after diagnosis on right parasternal long axis view: the left ventricle is less than one third of the right ventricle, left and right atria are dilated and the tricuspid valve is displaced because the aneurismal bulge, which is located of the upper part of the right ventricle.
An. aneurysm. Ao: aorta. ASD: atrial septal defect. LA: left atrium. LV: left ventricle. RA: right atrium. RV: right ventricle.

2A




2B




2C

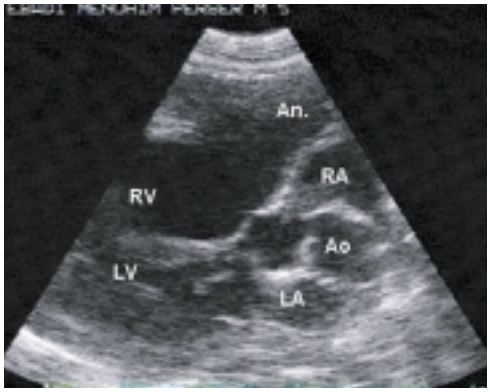
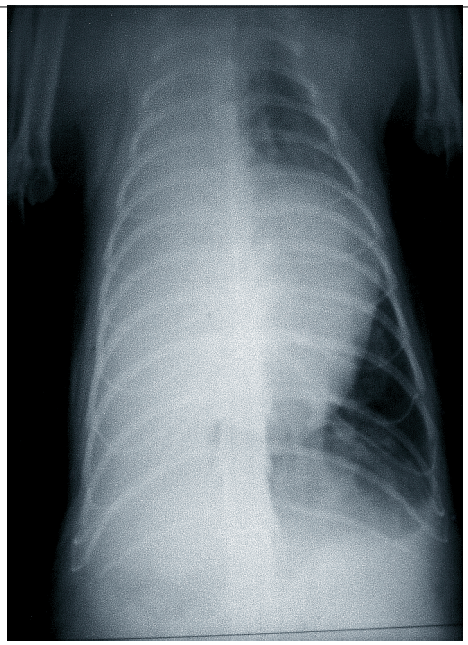
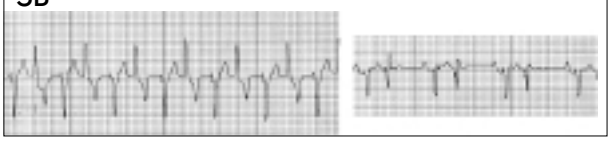



**Figure 3 - 3A.** Dorsoventral thoracic radiograph after 42 months showing a right sided pleural effusion. Lead II electrocardiogram (25 mm/sec., 1 mV/10 mm) after 42 months. **3B.** Permanent ventricular bigeminy.

3A



3B



considerably smaller than the maximal diameter.[7]

In our case, hypertrophic cardiomyopathy and ARVC were excluded by the clinical, echocardiographic and the pathological findings. The histological findings in the aneurysm were different from those observed in aneurysms related to an ARVC, where fatty cells replace myocardial cells and where fibrosis extends in a "wave front pattern" from the epicardium to the endocardium. A pseudoaneurysm was excluded by the presence of myocardial cells in the wall of the aneurysm and by its broad neck.[6,7]

The presence of a primary ASD was severe enough to create a volume overload of the right ventricle, eccentric hypertrophy and intramural myocardial changes. The unilateral right-sided pleural effusion, observed clinically, was likely due to congestive heart failure. In man, unilateral right-sided effusions are twice as common (28%) as left-sided effusions (15%) and bilateral effusions are seen in nearly 60% of the patients in chronic heart failure.[7] Unilateral effusions are also associated with pericardial disease.[8] As fibrous adherences were observed between the pericardial sac and the right atrium in our case, pericardial disease could also have contributed to the effusion. On necropsy, there were no signs of pulmonary hypertension. The slight interstitial fibrosis in the lungs could be due to a chronic congestion or the consequence of any resolved inflammation. The pulmonary edema was interpreted as a sign of excessive pulmonary blood flow (increased pulmonary capillary pressure or "overperfusion") and

reduced lymphatic drainage (lymphatic insufficiency to remove liquid from the interstitial space).

Clinically, classic auscultatory findings in cats with ASD are a soft left basilar systolic heart murmur and a split second heart sound due to the asynchronic closure of semilunar valve, as the right ventricular ejection time is prolonged[9] because of the increase of the amount of blood that the right ventricle has to eject. The auscultable heart murmur is not due to the flow across the ASD (physiologic pressure gradient in cats between LA and RA are approximately 6 mmHg in systole and 4 mmHg in diastole[10]), and large defects result in a large shunt with low velocity, which is normally not audible. Systolic turbulence is created by the increased blood flow through the pulmonic valve, and diastolic turbulence, normally silent in ASD, by the increased blood flow through the tricuspid valve. In this patient, the diastolic component of the murmur was also audible.

Regarding the therapy, the use of ACE-inhibitors was supposed to reduce the hypertrophic response to hemodynamic overload. In cats with primary pressure overload (pulmonary artery banding[11]), pharmacological modulation of the renin-angiotensin-aldosterone-system (RAAS) with captopril did not change the extent of hemodynamic overload and hypertrophic response, but the hypotheses that the volume overload of the right ventricle

**Figure 4 -** Lead II electrocardiogram (25 mm/sec., 1 mV/10 mm) after 43 months: development of multifocal premature contractions.

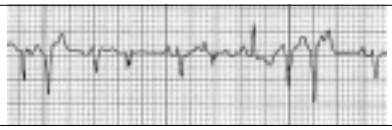


**Figure 5 -** Heart, after fixation in formaldehyde, showing the eccentric hypertrophy of the right ventricle, the atrium and the aneurysm. As the thin wall collapsed after fixation, surgical threads were placed in it and straight to demonstrate the aneurysm.
An. aneurysm. ASD: atrial septal defect. LV: left ventricle. RA: right atrium. RV: right ventricle.

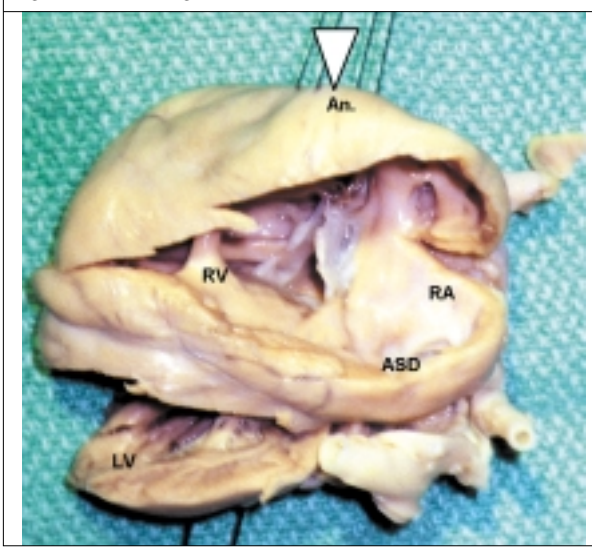
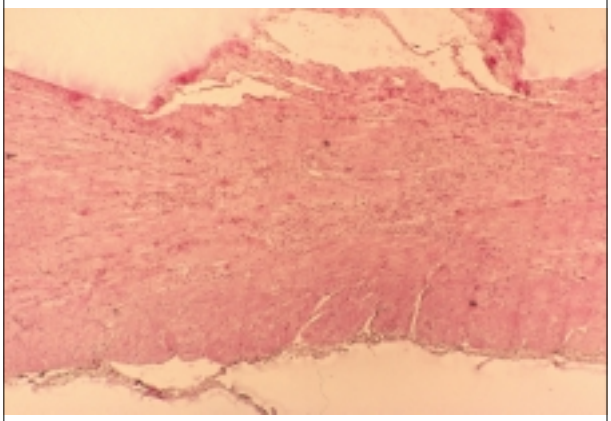
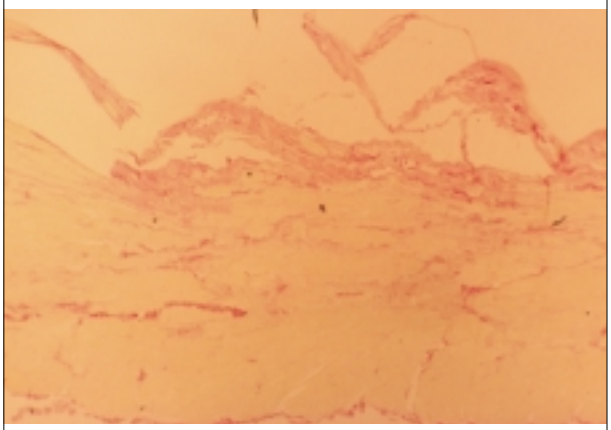



**Figure 6 -** Histopathology.
**6A.** wall of the right ventricle aneurysm. Endocardial and myocardial layers were thinner and partially replaced by fibroblastic and fibrotic tissue.
Discrete myocarditis (Hematoxylin-eosin stain; magnification x 40).
**6B.** wall of the right ventricle aneurysm. Fibrosis with collagen deposition mainly in endocardium and myocardium (Van-Gieson elastica stain; magnification x 40).
**6C.** detail of the myocardium from figure 6A. Fibroblastose/fibrocytose and discrete myocarditis in the wall of the aneurysm (Hematoxylin-eosin stain; magnification x100).

6A



6B



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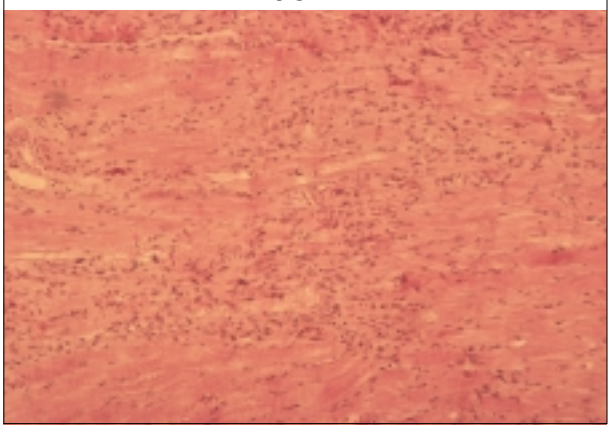

was related with chronic RAAS activation in this patient was most probable and benazepril was used because it was, on that time, the only ACE-inhibitor tested in cats.[3]

The antibiotic choice for the treatment of upper respiratory tract infection was done because of the difficulty for the cat owner to administer pills more than once a day. For the same reason, it was impossible to use selective nephron blockade with thiazide or spironolactones for the diuretic regimen.

The survival time of this patient was quite long, certainly over what had been expected at the first clinical examination. This case report shows that, even in presence of severe cardiac modifications or remodelling secondary to congenital disease at a young age, clinicians should be prudent when giving short time prognosis. In this cat, chronic occurring upper airways disease was probably more important for the impaired quality of life as the heart problem.

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