

Aortic dissecting aneurysm associated with systemic arterial hypertension in a cat

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Summary

Aortic dissection is characterized by a tear in the aortic wall resulting in blood from the aortic lumen penetrating into the media, which causes dissection. When aortic dissection does not cause rupture, it provokes localized dilation of the aorta or aneurism, also called dissecting aortic aneurism (DAA). This case report describes a DAA in a cat associated with systemic arterial hypertension (SAHT). A 10-year-old male Domestic shorthair cat was presented for cardiac evaluation. Anamnestic clinical complaints were a syncope associated with paraparesis and weak femoral pulses. Cardiomegaly had been found radiographically, and cardiogenic arterial thromboembolism had been suspected. Upon presentation physical abnormalities were tachycardia and a heart murmur. Measurement of systolic systemic arterial blood pressure (SABP) revealed severe SAHT. Echocardiographic images showed severe DAA, and marked aortic valve insufficiency. Palliative antihypertensive treatment resulted in fast clinical improvement and significant decrease in blood pressure. Four months later, acute severe respiratory distress due to cardiogenic pulmonary edema led to the cat's euthanasia. In human medicine, DAA is a well-reported complication of SAHT. This is the second case of DAA with congestive heart failure reported in a hypertensive cat.

Keywords: heart, feline, blood pressure, aorta, aneurysmal dilation

Aneurysma dissecans aortae in Verbindung mit systemischer Hypertension bei einer Katze

Die Aortendissektion ist durch einen Riss in der Aortenwand charakterisiert, der dazu führt, dass Blut aus dem Aortenlumen in die Tunica media eintritt. Dies verursacht eine Aufspaltung der arteriellen Wandschichten. Wenn die Aortendissektion keine Ruptur verursacht, führt sie zu einer lokalen Dilatation der Aorta oder einem Aneurysma, auch Aneurysma dissecans aortae oder dissecting aortic aneurism (DAA) genannt. Dieser Fallbericht beschreibt ein DAA in Verbindung mit einer systemischen arteriellen Hypertension (SAHT) bei einer Katze. Eine zehn Jahre alte männliche Europäische Kurzhaar Katze wurde für eine kardiologische Untersuchung vorgestellt. Die Anamnese ergab eine Synkope sowie eine Paraparese und schwachen femoralen Puls. Kardiomegalie wurde radiographisch nachgewiesen und kardiogene arterielle Thromboembolie wurde vermutet. Bei der klinischen Untersuchung waren Tachykardie und ein Herzgeräusch feststellbar. Die Messung des systemischen arteriellen Blutdrucks (SABP) zeigte eine schwere SAHT. Echokardiographische Bilder wiesen ein DAA nach, in Verbindung mit einer deutlichen Aortenklappeninsuffizienz. Palliative antihypertensive Therapie führte zu einer schnellen klinischen Besserung sowie deutlicher Reduktion des Blutdrucks. Vier Monate später wurde die Katze wegen akuter und schwerer Atemnot aufgrund eines kardiogenen Lungenödems euthanasiert. In der Humanmedizin ist das DAA eine bekannte Komplikation arteriellen Bluthochdrucks. Dies ist der zweite publizierte Fall von DAA mit kongestiver Herzinsuffizienz bei einer hypertensiven Katze.

Schlüsselwörter: Herz, Katze, Blutdruck, Aorta, Aneurysmal Dilatation

Introduction

Aortic dissection is a rare, severe, acquired complication elicited by a primary tear in the intima resulting in blood from the lumen entering the diseased media, leading to dissection and creating a true and false lumen (Braverman et al., 2012; Sheikh et al., 2013). Another hypothesis is that a primary rupture of the vasa vasorum leads to hemorrhage in the aortic wall, with subsequent intimal disruption, creating the intimal tear and aortic dissection (Braverman et al., 2012; Sheikh et al., 2013). In humans, aortic dissection is usually secondary to another systemic disease, such as systemic arterial hypertension, an abnormality of connective tissues, or a parietal trauma (Braverman et al., 2012; Sheikh et al., 2013). When aortic dissection does not cause rupture, it provokes localized dilation of the aorta or aneurysm, also called dissecting aortic aneurysm (DAA).

History and clinical presentation

A 10-year-old castrated male Domestic shorthair cat was referred to the Alfort Cardiology Unit for cardiovascular exploration. A heart murmur had been detected 6 months earlier by the referring veterinarian and a treatment with benazepril (Fortekor[®], 0.5 mg/kg SID *per os*) was prescribed. Five months later, the cat was presented for syncope associated with tachypnea, paraparesis, and weak femoral pulses. Thoracic radiographs revealed cardiomegaly with no radiographic evidence of pulmonary edema. Treatment with nadroparin (Fraxiparine[®], 100 UI/kg SID, subcutaneously) and salicylic acid (Aspégic nourrisson[®], 5 mg/kg every 72 hours, *per os*) was initiated by the same veterinarian. Symptoms

resolved within 24 hours, nadroparin was then discontinued, but benazepril and salicylic acid were continued. At presentation, auscultation revealed tachycardia (210 bpm), a systolic-diastolic discontinuous grade IV/VI left basal heart murmur, and a systolic grade IV/VI left apical heart murmur. The rest of the physical examination was unremarkable.

Echocardiographic findings and interpretation

An echocardiographic examination was performed using a Vivid 7 (Dimension BT04 digital ultrasound system, General Electric medical system, Waukesha, WI, USA) equipped with 7S (3.5-8 MHz) and 10S (4.0-11.5 MHz) phased-array transducers with continuous ECG monitoring. Two-dimensional echocardiography showed a marked dilation of the aorta, and a membranous structure parallel to the aortic wall, originating from the right base of the aorta, extending through the entire visible portion while bulging into the aortic lumen (Fig. 1). Color-flow Doppler mode revealed two aortic systolic flows separated by the membranous structure, i.e., a laminar flow (blue) within the anterior part of the aorta and a turbulent flow posteriorly (Fig. 2). Continuous-wave Doppler mode confirmed an increased peak systolic aortic flow velocity (2.79 m/s, reference range [0.8-1.9], Chetboul et al., 2006), which would explain the systolic left basal heart murmur. Additionally, severe holodiastolic aortic regurgitation, with a short pressure half-time (78 ms, Boon, 2011), was noted explaining the diastolic basal heart murmur (Fig. 3). The aortic regurgitation was also characterized by an early-diastolic peak velocity of 7.85 m/s as assessed by

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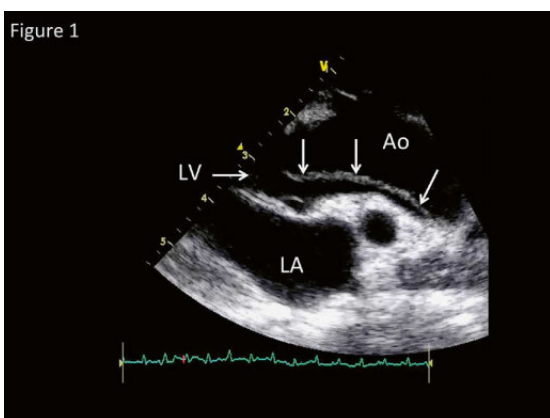


Figure 1: Two-dimensional echocardiogram: right parasternal long-axis view of the aorta. Note the marked dilation of the aorta, as well as the thin-walled membranous structure (arrows) that originates from the aortic base and extends through all its visible part. Ao: aorta; LA: left atrium; LV: left ventricle.

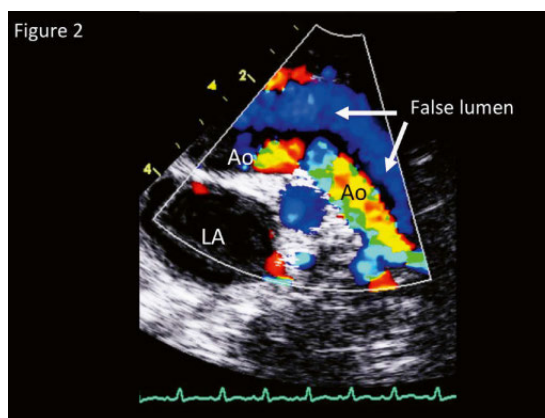


Figure 2: Color-flow Doppler mode echocardiogram obtained from the right parasternal long-axis view of the aorta. Note the two parallel blood flows within the aorta, i.e., one of low velocity (without any aliasing artifact) in the dissecting aneurysm (blue) and one aliased turbulent flow under the membranous structure. Ao: aorta; LA: left atrium.

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Table 1: Two-dimensional and M-mode variables obtained in a 10-year-old DSH cat suffering from dissecting aortic aneurism. Normal ranges were established from a population of 100 healthy cats (Chetboul et al., 2006).

Variables	Value	Normal ranges
Left atrium/aorta ratio at end-diastole	1.1	0.6 – 1.1
Left ventricular end-diastolic diameter (mm)	20.6	10.1 – 18.4
Left ventricular end-systolic diameter (mm)	11.2	3.5 – 10.8
Interventricular end-diastolic septum (mm)	5.1	3.4 – 5.6
Interventricular end-systolic septum (mm)	8.3	5. – 8.8
Left ventricular end-diastolic free wall (mm)	4.8	2.7 – 5.2
Left ventricular end-systolic free wall (mm)	9.2	5.1 – 9.1
Fractional shortening (%)	45.8	29 – 69

continuous-wave Doppler mode (Fig. 3), with a corresponding estimated mean arterial pressure of at least 246 mmHg using the modified Bernoulli equation ($4 \times 7.85^2 = 246$ mmHg). Left ventricular eccentric hypertrophy with a normal shortening fraction was also observed (Table 1, Chetboul et al., 2006). Mild mitral valve regurgitation resulting from dilation of the mitral valve annulus was also noted explaining the systolic apical heart murmur. Owing to the combination of high-velocity aortic regurgitation, aortic root dilation and left ventricular remodeling, systemic arterial hypertension associated with a DAA complication was suspected.

Treatment and outcome

Indirect systemic arterial blood pressure (ABP) was measured by Doppler technique (811-BL, Parks Medical Electronics Inc, Aloha, Ore) and confirmed major systemic systolic hypertension (systolic ABP = 260 mmHg,

Brown et al., 2007). The whole blood count and biochemical analysis were unremarkable, except for mild hypokalemia ($K^+ = 3.1$ mmol/L, reference ranges [3.6-5.5]). The owners refused any further exploration of the hypertension causes. An antihypertensive treatment with lercanidipine was initiated (Lercan[®], 0.79 mg/kg SID, *per os*). Administration of benazepril and salicylic acid was continued.

A control was performed 10 days later. The ABP was significantly lower (systolic ABP = 180 mmHg). Clinically the cat was doing well, pulse quality was considered normal, and the systolic and diastolic components of the heart murmur decreased in intensity.

Four months after the initial presentation, the cat was presented to the referring veterinarian for sudden respiratory distress. Thoracic radiographs revealed severe pulmonary edema. The owners refused further treatment, opted for euthanasia and declined necropsy evaluation.

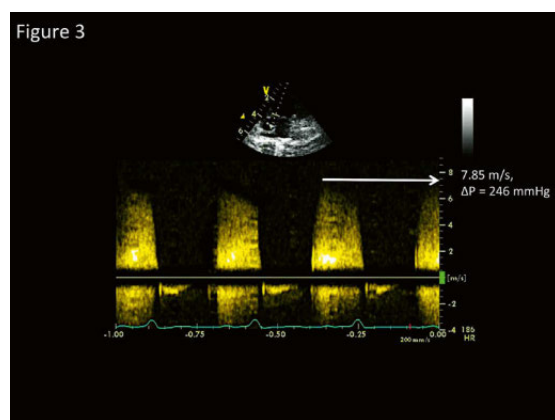


Figure 3: Continuous-wave Doppler mode recorded at the level of the aorta from the left apical 5-chamber view. Note the high early-diastolic flow velocity (maximal velocity = 7.85 m/s,) associated with a rapid decline (pressure half time of 78 ms), respectively consistent with increased mean aorta-left ventricle pressure gradient (246 mmHg) and severe aortic insufficiency.

Discussion

The clinical presentation and echocardiographic findings in this case were typical of systemic arterial hypertension with DAA and very similar to those reported in humans (Braverman et al., 2012; Sheikh et al., 2013). However, the initial clinical presentation could also be consistent with aortic thromboembolism, which is a common complication of feline cardiomyopathies (Smith et al., 2003; Borgeat et al., 2014).

Dissecting aortic aneurism is an extremely rare condition and, to the best of our knowledge, this is only the fifth case of DAA described in the feline species: 2 of them in association with systemic arterial hypertension (Wey and Atkins, 2000; Scollan and Sisson, 2014), 1 with endocarditis (Lourenco et al., 2002), and 1 of unknown etiology in a diabetic cat (Newhard and Jung, 2017). Sporadic cases have also been reported in dogs

related to a suspected elastin dysplasia comparable to the human Marfan syndrome (3 dogs, Boulineau et al., 2005; Lenz et al., 2015), to obstructive aortic chondrosarcoma (1 dog, Cohen et al., 2010), to patent *ductus arteriosus* (PDA) with the dissection going from the pulmonary artery through PDA into aorta (Jenni et al., 2007), and to non specific or unidentified causes (3 dogs, Belivilacqua et al., 1981; Waldrop et al., 2003; Cornelis et al., 2014).

The clinical signs associated with DAA in small animals are similar to those described in humans: syncope, pain, symptoms of congestive heart failure and neurological signs (Braverman et al., 2012; Sheikh et al., 2013). Hypoperfusion of the posterior limbs, as described in humans suffering from DAA, may have accounted for the paraparesis observed at initial presentation of our case. Similarly, the syncope reported one month earlier could be due, at least in part, to a dull pain at the time of the dissection, as described in human patients (Braverman et al., 2012; Sheikh et al., 2013). Diagnosis in humans is usually achieved by computed tomography and transesophageal echocardiography (Braverman et al., 2012; Sheikh et al., 2013; Sun et al., 2014). Imaging techniques such as magnetic resonance imaging, aortography and echocardiography may also be employed as was the case here (Braverman et al., 2012; Sheikh et al., 2013). In veterinary medicine, echocardiography is very useful for the non-invasive diagnosis of DAA in awake animals (Wey and Atkins, 2000; Lourenco et al., 2002; Waldrop et al., 2003; Cohen et al., 2010), providing highly evocative images as in this report.

In the present case, the cause of the DAA was considered to be systemic arterial hypertension. This is also a well-recognized predisposing factor in humans, where hypertension is usually primary, associated with vascular disease (Braverman et al., 2012; Sheikh et al., 2013). Investigation for underlying causes of systemic arterial hypertension was declined by the cat's owners. However hyperaldosteronism could be suspected based on the hypokalemia detected during the initial presentation (Reusch et al., 2010).

In humans, treatment of DAA depends on whether the condition is acute or chronic and on the exact site of the dissection. According to Stanford classification of aortic dissection, type A dissections involve the ascending aorta, with or without extension into the descending aorta, and type B dissections are those that do not involve the ascending aorta (Braverman et al., 2012). The early mortality rate in case of acute aortic dissection is very high, with a mortality rate up to 1% to 2% per hour reported in the first hours. Emergency surgery improves survival in acute type A dissections, whereas initial medical therapy is recommended for acute type B dissections. The medical management of aortic dissections aims at stabilizing the patient, controlling pain, lowering blood pressure, and reducing the rate of rise and force of left ventricular ejection (Braverman et al., 2012). In our case, the treatment consisted of antihypertensive and antithrombotic drugs together with an angiotensin converting enzyme inhibitor. Surgery, although not described in the veterinary literature, could have been an additional option.

The prognosis of DAA in small animals has not been examined thoroughly, although some cats may tolerate the aneurism for several months (Wey and Atkins, 2000; Scollan and Sisson, 2014). In our case the animal was euthanized four months after initial presentation, owing to severe congestive heart failure rather than to rupture of the DAA.

In conclusion, DAA, although rare, needs to be taken into account as a possible secondary complication when investigating cats with systemic arterial hypertension and also as a differential diagnosis of acute paresis.

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