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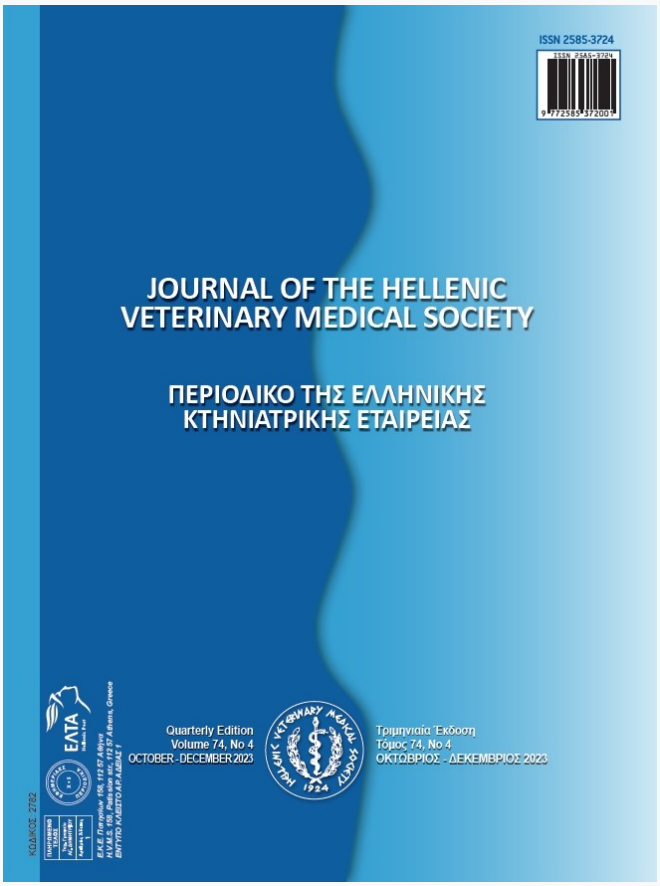

Synovial Hemangioma of the Stifle Joint in a Kangal Dog

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Case report
Κλινικό περιστατικό

Thoracic and abdominal aortic dissection ina hypertensive hyperthyroidcat

C. Veciana Basse, I. Testault

CHV Atlantia

Abstract: Aortic dissection is an uncommon finding in both dogs and cats. The precise pathophysiological mechanisms responsible for the development of aortic dissection remain unclear. 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, with chronic systemic hypertension being the most common cause. Although systemic hypertension is not uncommon in geriatric cats, aortic dissection remains a rare disease, and very few case reports including post-mortem examination are available to date. This is a case of an aortic dissection in a hypertensive hyperthyroid cat, presenting with haemorrhagic pericardial effusion. The dissection affected both thoracic and abdominal aorta, with histopathological confirmation. This case is an example of the utility of both thoracic and abdominal ultrasound for the diagnosis of aortic dissection in cats and it highlights the importance of rigorous clinical examination. Further investigations are fundamental to understand how dissections develop and extend to achieve good clinical results. Although the association between aortic dissection and pericardial effusion remains unclear, aortic dissection should be part of the differential diagnosis in a cat presenting with pericardial effusion.

Aortic dissection should be considered in the differential diagnosis of pericardial effusion in cats.

Keywords: aorta; dissection; pericardial effusion; cat.

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CASE DESCRIPTION

A 13-year-old spayed female domestic short haired cat was referred for evaluation of cardiomegaly and pleural effusion based on thoracic radiographs obtained prior to referral. The cat had a history of lethargy and acute dyspnoea for two days. On physical exam, the cat weighed 4. 8kg and body condition score was 4/9. The cat was lethargic, tachypnoeic, dyspnoeic and presented a restrictive breathing pattern. The mucous membranes were pink and moist and capillary refill time was within normal limits. No mucous lesions or bleedings were observed. Jugular veins were distended. Thyroid glands were bilaterally enlarged. Regular strong symmetric femoral pulses were palpable bilaterally and heart rate was 260 bpm. Heart sounds were muffled bilaterally and no murmur or any other abnormal heart sound was detected. Rectal temperature was normal. The cat was admitted, and the first diagnostic tests were performed.

The thoracic FAST scan revealed a large volume pericardial effusion causing cardiac tamponade. In addition, there was a moderate volume pleural effusion (Figure 1).

Blood biochemistry was performed - urea and creatinine levels were within their reference intervals (urea 11. 3 mmol/L, reference 3. 3-11. 65 mmol/L; plasma creatinine concentration 58 μmol/L, reference 53-176 μmol/L). Coagulation times were within acceptable limits: Prothrombin Time (PT) was equal to17 seconds (feline reference range 15-22 seconds); Partial Thromboplastin Time (PTT) was equal to 99 seconds (feline reference range 50-115 seconds).

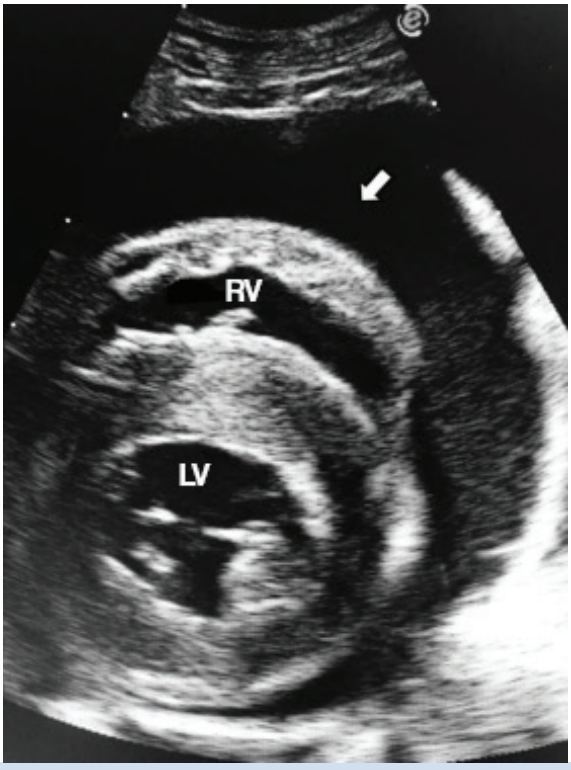

Figure 1. Right parasternal 2-dimensional short-axis echocardiography revealing pericardial effusion (white arrow). The left ventricle (LV) and the right ventricle (RV) are identified.

EMERGENCY TREATMENT AND FOLLOW-UP

Ultrasound guided pericardiocentesis was performed, and 20 millilitres of a haemorrhagic fluid were removed. After pericardiocentesis, treatment for

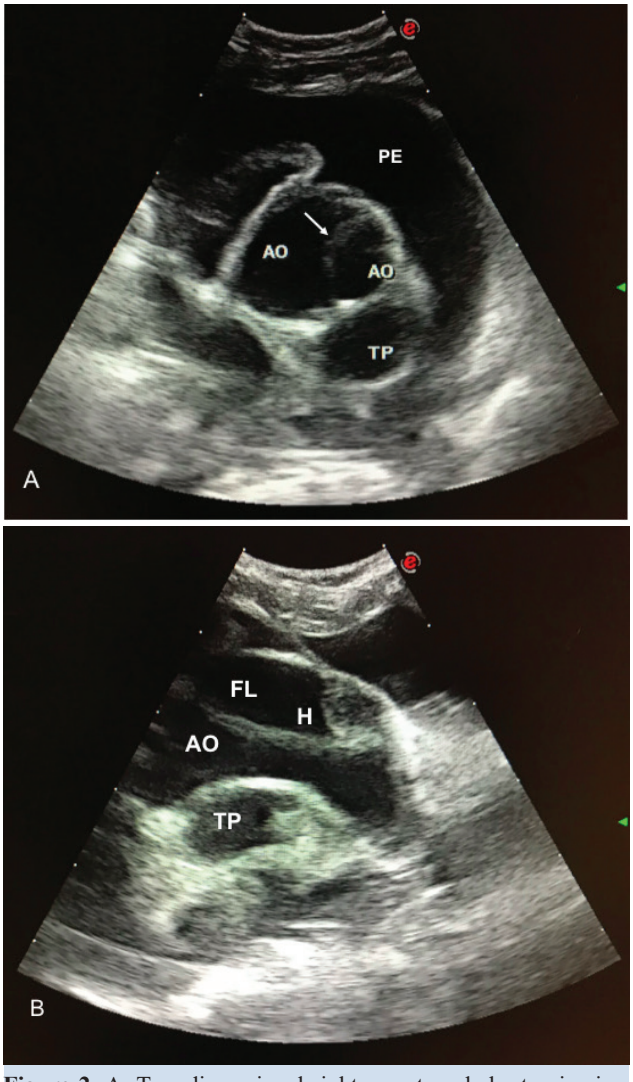

Figure 2. A. Two-dimensional right parasternal short axis view demonstrating a dissecting flap (white arrow) separating the Aorta in two different chambers. Pericardial effusion is noted. B. Longitudinal view of thoracic Aorta. The dissection is separating the Aorta in two distinct chambers, the true lumen (Aorta) and the false lumen (FL). A hematoma is identified within the false lumen. AO: aorta; TP: Main Pulmonary Artery; PE: pericardial effusion, FL: false lumen, H: hematoma.

suspected acute congestive heart failure was initiated using diuretics (2 mg/kg IV SID: Furosemide, Dimazon®, MSD Animal Health), and oxygen therapy.

Twenty-four hours after initial presentation, a complete cardiac ultrasound was performed.

The presence of a membrane parallel to the aortic wall was observed in both cardiac and abdominal ultrasound. The membrane showed two separate aortic chambers, highly suspicious of aortic dissection, extending from the ascending Aorta to the abdominal Aorta (Figure 2 and Figure 3). Colour-flow Doppler revealed two distinct aortic systolic flows separated by the dissection. The flow was laminar into the true lumen of the aorta, even though it was narrowed 2. A recurrent pericardial effusion was noted. Two-dimensional right parasternal short axis view at the level of the *chordate tendinae* allowed diastolic measurements of interventricular septum (IVSd), in multiple locations,and of left ventricular free wall (LVFWd) thicknesses; the resulting measurements were as follows: IVSd=7-7. 5 mm; LVFWd=6. 8mm. Two-dimensional right parasternal short axis heart base view allowed end-diastolic measurements of aortic (Ao) and left atrial (LA) diameter for calculation of LA/Ao ratio, which was equal to 1. 5. According to Luis Fuentes *et al* (2020)[9], these measurements suggested diffuse thickening of interventricular septum and left ventricular free wall and left atrial chamber enlargement. No thrombus was present within the left atrium.

Cytological analysis was performed on the pericardial fluid; the results were consistent with haemorrhagic effusion containing mesothelial cells without any sign of malignancy and with evidence of mild inflammation.

Hematologic and biochemical evaluation revealed a mildly increased activity of hepatic enzymes and mild hyperglycaemia, the latter being compatible with a stress response. The rest of the biochemistry and the results of the complete blood count were unremarkable. The total serum thyroxine (TT4) concentration was markedly elevated consistent with hyperthyroidism (Table 1).

Twenty-four hours after initial presentation, systolic blood pressure was measured by Doppler technique (811-B, Parks Medical Electronics Inc, Aloha, Oregon). Five measurements were performed. Systemic systolic hypertension was confirmed with a final average result of 230 mmHg, with the lowest measurement at 225 mmHg and the highest measurement at 235 mmHg.

Given the guarded prognosis, further evaluation was declined by the cat's owners and medical management and supportive care were requested. Seventy-two hours after initial presentation and no improvement of clinical signs, humane euthanasia was elected.

DIAGNOSIS CONFIRMATION

The owner consented to a post-mortem examination, which confirmed an abnormality in the aortic wall extending distally from one of the aortic leaflets to the abdominal aorta (Figure 4). This lesion consisted of the rupture of the tunica intima associated with the formation of a pseudo cystic cavity within the tunica media, surrounded by fibroblastic tissue. The histopathological analysis report confirmed observation of an aortic wall dissection.

Histologic evaluation of the heart, kidneys and thy-

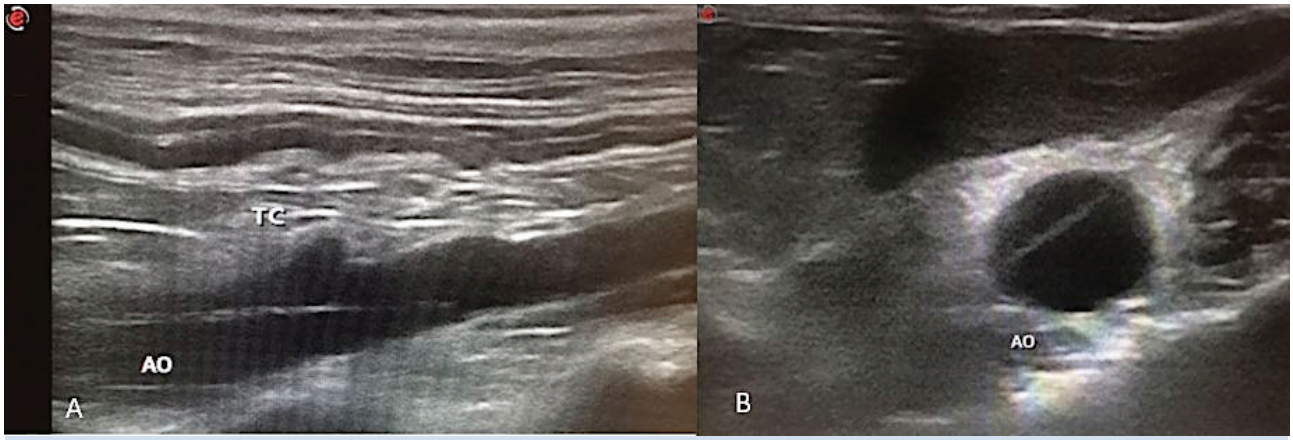

Figure 3. Longitudinal and transverse sonographic images of abdominal (A) and thoracic (B) aorta.An intimal flap is identified delineating the true lumen of the aorta (Ao) from the false channel. TC: celiac trunk.

Table 1. Complete blood tests results

Parameter	Result	Reference range
Biochemistry		
ALP	241 IU/L	20-190 IU/L
ALAT	167 IU/L	8-100 IU/L
Glucose	154.8 mg/dL	59.94-149.4mg/dL
Alb	29 g/L	24-37 g/L
Urea	28.85 mg/dL	9.24- 32.63 mg/dL
Creatinine	81 μmol/L	53-176 μmol/L
Haematology		
WBC	6.3 x 10^9/L	5-11 x 10^9/L
Lymphocytes	1.1 x 10^9/L	1-4 x 10^9/L
Monocytes	0.1 x 10^9/L	0-0.5 x x 10^9/L
Granulocytes	5.1 x 10^9/L	3-12 x 10^9/L
Eosinophils	0.06 x 10^9/L	0-6 x 10^9/L
RBC	8.26 x 10^{12}/L	5-10 x 10^{12}/L
Ht	35.7 %	27-47 %
Hb	109 g/L	80-170 g/L
MCV	43 fL	40-55 fL
PLT	384 x 10^9/L	180-430 x 10^9/L
Serum Total Thyroxine		
TT4	189 nmol/L	10-60 nmol/L

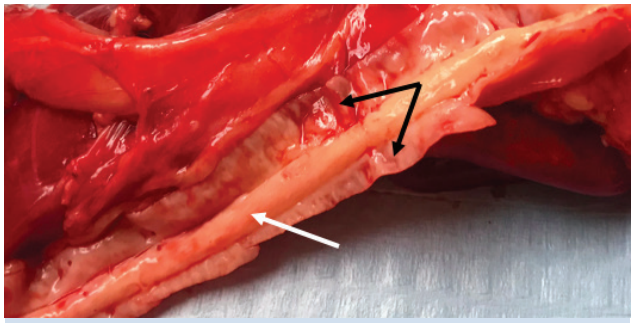

Figure 4. Macroscopic examination of the thoracic aorta. Aorta (white arrow) is surrounded by the false lumen (black arrows). The latter has been opened all along.

roid glands were performed. There was evidence of bilateral thyroid adenomas. There was also epicarditis consistent with an inflammatory response secondary to the pericardial effusion. The presence of a circumscribed region of clotted blood, fibrinous material and a moderate fibrosis, resulting in irregular epicardial thickening, were also observed. Renal abnormalities consistent with chronic multifocal tubulointerstitial nephritis and associated glomerulonephritis were also noted. The macroscopic and histological renal abnormalities described did not exclude a hypoxia-mediated response, such as kidney infarction. Left ventricular concentric hypertrophy was not confirmed on histological evaluation.

DISCUSSION

Aortic dissection is an uncommon finding in both dogs and cats. The precise pathophysiological mechanisms responsible for the development of aortic dissection remain unclear. [13] Nowadays, it is thought that an underlying disease is necessary for the formation of an aortic dissection. The integrity of the aortic wall can also be diminished by age. [16] Another hypothesis is that a primary rupture of the vasa vasorum leads to haemorrhage in the aortic wall, with subsequent intimal disruption, creating the intimal tear and aortic dissection. [2,6] In humans, aortic dissection is usually secondary to another systemic disease, such as systemic arterial hypertension, an abnormality of connective tissues (such as in Marfan's syndrome), or a parietal trauma. [2,6]

Aortic dissection is characterized by a primary tear in the arterial intima, resulting in acute leakage of blood from the lumen into the aortic wall, penetrating the intima and entering the media, dissecting its layers. A flap can then develop, separating the true aortic lumen from the blind pocket (also referred to as false lumen or false channel). The dissection can occur into any point of the thoracic aorta (ascending aorta, aortic arch or descending aorta) and can extend proximally or distally (or both) from its site of origin. As reported by D'Ancona *et al*(2014)[5], haemodynamics after aortic dissection are affected by the aortic anatomy and tear morphology. In human medicine, the Stanford

classification defines types A and B depending on the site of origin of the dissecting flap. In Stanford type B, the ascending aorta and the proximal aortic arch are spared and the dissection initiates from the descending aorta. [13] The false lumen can also have a proximal entry, a distal re-entry or both (in the latter case called patent false lumen). Qing *et al* (2012)[13] demonstrated that distal re-entries were associated with lower pressure in the false channel and the high intra-aortic pressure splits the media in a laminar fashion. As reported by Waldrop *et al* (2003)[14], pulsatile ventricular flow promotes progression of the dissection. Aortic dissection can include severe complications as stroke, aortic valve insufficiency, cardiac tamponade and aortic rupture. [15]

In this case, a systemic hypertension due to hyperthyroidism was thought to be the underlying cause of the dissection. Oricco *et al* (2019)[10], described a case series of aortic dissection in four cats; however, at presentation, all cats had normal blood pressure measurements. In the case reported herein, consistently increased blood pressure measurements strongly supports underlying systemic hypertension. In human patients, aortic dissection can be associated with both hypotension, in case of proximal dissection, and hypertension, more common when distal dissections occur[10]. In this case, the dissection was both proximal and distal, extending from one of the aortic leaflets to the abdominal aorta. Echocardiography was consistent with interventricular septum and left ventricular free wall thickening, suggesting concentric left ventricle hypertrophy secondary to increased after load (ie. systemic hypertension). Left atrial chamber enlargement strongly suggested congestive heart failure, which was believed to be responsible for the acute onset of clinical signs in the case described herein. However, histopathological examination did not reveal the typical features of hypertrophic cardiomyopathy. Sonographically observed left ventricular concentric hypertrophy could have been related to pseudohypertrophy due to reduced preload, caused by the cardiac tamponade. Pleural effusion had been attributed to the congestive heart failure. Biasato *et al* (2017)[1] have recently described a case of pulmonary artery dissection resulting in a haemothorax in a cat. In the case described herein, only the pericardial fluid resulting in tamponade was analysed, and whether the pleural effusion was haemorrhagic or not remains unknown. A small blood clot was identified at post-mortem examination adjacent to the epicardium surface, along with fibrinous material. It is likely this circumscribed lesion was already present at the time of presentation and had not been identified during echocardiography examination. The hypothesis that this material developed after echocardiography examination seems less likely. It remains unclear whether the haemorrhagic pericardial effusion in this cat was a consequence of the aortic dissection itself. Interestingly, out of the four cases reported by Oricco*et al* (2019)[10], three cats presented with acute onset of clinical signs and concurrent pericardial effusion resulting in cardiac tamponade. Further investigations are needed to evaluate the association between pericardial effusion and cardiac tamponade with aortic dissection in cats. The obstruction of the visceral branches by the dissecting flap can cause organ malperfusion by ischemia, as well as paraparesis or paraplegia secondary to deranged spinal blood flow or femoral arterial emboli.[14] In the case described herein, the dissection was identified all along the thoracic aorta and a portion of the abdominal aorta. According to Qin *et al* (2016)[12], acute aortic dissection is also characterized by systemic inflammatory response syndrome (SIRS), which may worsen the aortic dissection and affect the prognosis. Sudden death can occur because of rupture of the vessel.

Treatment of aortic dissection in humans depends on whether the condition is acute or chronic and on the exact site of the dissection[2,6] (according to Stanford classification previously mentioned). The medical management of aortic dissections aims at stabilizing the patient, controlling pain, lowering blood pressure, and reducing the force of left ventricular ejection. [3]Oricco*et al*(2019)[10] report one of the cats in their series was started on nitroglycerine and furosemide, along with oxygen therapy, after echocardiographic diagnosis of aortic dissection. The treatment allowed improvement of symptoms and the cat was discharged on furosemide and enalapril. The same cat presented four years after initial presentation with clinical signs and radiographic images consistent with arterial thromboembolism and cardiogenic pulmonary oedema. Although no post-mortem examination could be performed, their long-term follow-up suggests a therapeutic plan composed of furosemide and enalapril should be considered in patients diagnosed from aortic dissection. According to Waldrop *et al (*2003)[14] acute aortic dissection is a clear contraindication for the use of antithrombotic drugs, given the inhibition of clotting mechanisms that potentially could stem further dissection. Liu *et al* (2016)[8] raised the possibility that preventive administration of

UDCA (antioxidant ursodeoxycholic acid) and other antioxidants to patients at risk could be useful, since oxidative stress plays an important role in the pathogenesis of many vascular diseases, including acute aortic dissection. 8, 4,7,11

Prognosis of aortic dissection is nowadays assumed to be poor in domestic animals. Medical and surgical treatments remain a big challenge because the underlying pathological mechanisms responsible for aortic dissection are still unclear.

CONCLUSION

This case is an example of the utility of ultrasound for the diagnosis of aortic dissection in cats and it highlights the importance of rigorous clinical examination. Further investigations are fundamental to understand how dissections develop and extend in order to achieve good clinical results. Post-mortem examination has rarely been performed in cats with aortic dissection. Although the association between aortic dissection and pericardial effusion and tamponade in cats remains unclear so far, the authors recommend considering aortic dissection in the differential diagnosis of pericardial effusion in cats.

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