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WHAT'S YOUR DIAGNOSIS?

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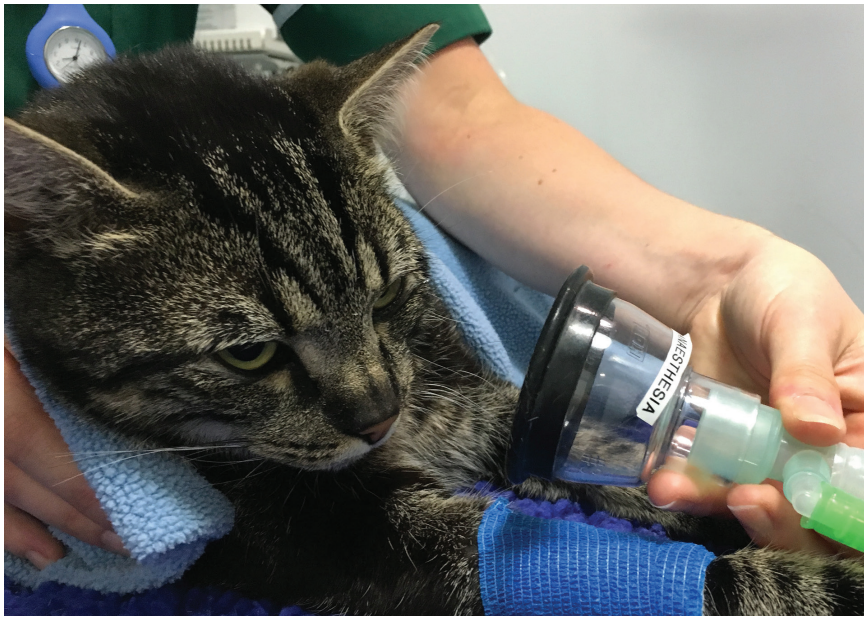
Case presentation.
A six year old, male neutered domestic shorthair was presented as an emergency out of hours with a history of reported sudden onset seizure-like activity (collapse and tonic/clonic movements), following being found under a neighbour's car. Previous to this the cat had been reportedly healthy, was fed commercial dry diet and had outdoor access. Routine vaccination, althelmintics and flea prevention were up to date.

Physical examination revealed the cat to be quiet, alert and aware and in average body condition (BCS 5/9). Heart rate was 240bpm, with poor peripheral pulse quality, mucous membranes were pale and a CRT of 1 second. There was marked expiratory dyspnoea and effort with a respiratory rate of 60 breaths per minute and open mouth breathing. Harsh lung sounds were noted caudodorsally bilaterally. Femoral pulses were palpable though deemed weaker than normal, metatarsal pulses were not palpable.



A six year old cat presented with reported seizure-like activity and marked dyspnoea.

What is your problem list at this stage?

Current problem list includes:

- *Expiratory dyspnoea*
- *Harsh caudodorsal lung sounds*
- *Tachycardia*
- *Pallor*
- *Single seizure episode*

Can you refine the problem list further?

- ***Expiratory dyspnoea*** may be primary lower airway airway disease (e.g. Feline allergic lower airway disease – asthma), pleural space disease (e.g. Pneumothorax/pleural effusion), pulmonary disease (e.g. non-cardiogenic pulmonary oedema, pneumonia, bronchopnuemopathies), disease involving the thoracic wall and/or diaphragm and secondary to pulmonary oedema (cardiogenic vs. non-cardiogenic).
- ***Harsh caudodorsal lung sounds*** may be associated to lower airway disease, pulmonary oedema (e.g. cardiogenic/non-cardiogenic – possibly secondary to prolonged seizure activity), parasitic lung disease (e.g. Aelurostrongylus abstrusus), and pulmonary contusions.
- ***Tachycardia*** may be primary cardiac disease (e.g. hypertrophic cardiomyopathy), or secondary including pain, stress, fear, anaemia or hypoxaemia.
- ***Pallor*** may be associated to anaemia or poor peripheral perfusion (e.g. cardiac failure, hypovolaemia).
- ***Seizure episodes*** may be classified as either extracranial (e.g. metabolic, toxic, traumatic or hypoxaemic) or intracranial (e.g. lesions causing intracranial pressure increases, or primary epilepsy).

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What would you do next?

The cat was placed in an oxygen cage and an IV catheter was placed. A venous blood sample was collected at time of IV placement. IV Butorphanol 0.2mg/kg and Furosemide 2mg/kg were given. After 20 minutes in 100% oxygen the respiratory rate had reduced to 44bpm, open mouth breathing had ceased and heart rate reduced to 210 beats per minute.

A minimum database was performed including PCV/TS, blood glucose and plasma lactate *(figure 1)* as well as well as full haematology and biochemistry *(figures 2 & 3)*.

How would you interpret these results?

Haematology revealed a leucocytosis with a mature neutrophilia, monocytosis and eosinopenia which could be explained as a stress leucogram, but infection/ inflammation and neoplasia are also possible causes. The haematology indicates a severe thrombocytopenia. At this level there is a significant risk of spontaneous haemorrhage, but if true rules out the possibility of haemorrhage as the cause of the thrombocytopenia. Biochemistry revealed a mild increase in BUN, indicating the cause is most likely pre-renal. The remainder of the biochemistry was unremarkable.

Hypoglycaemia as an underlying cause of the seizure like activity was not evident. A normal HCT and erythrocyte panel also makes haemorrhage and anaemia unlikely as a cause for this cat's pallor; poor perfusion is a possible explanation.

What would you do now?

A manual blood smear was performed to further assess the thrombocytopenia. This was assessed to be an absolute thrombocytopenia with ≤1 platelet observed per high power field. The feathered edge revealed no platelet clumping. Pulse oximetry revealed a saturation of haemoglobin of 93%. Non-invasive Doppler blood pressure was 80mmHg. The cat's condition had improved following oxygen therapy and IV Furosemide, however remained tachypnoeic with a respiratory rate of 52bpm. Further IV Furosemide 2mg/kg was given, reduced to 1mg/kg every 4 hours and the cat remained in an oxygen-rich kennel for the duration of the night. Due to the risk of stress induced desaturation thoracic radiographs were not performed at this stage.

The following morning...

Following treatment and oxygen therapy overnight the cat's respiratory rate and effort was 40 breaths per minute and the harsh caudo-dorsal lung sounds had reduced. Heart rate was 218 beats per minute. A gallop rhythm and jugular distention were now noted on clinical examination. Neurological evaluation of the cat was unremarkable. Conscious thoracic radiographs were attempted now the cat was more respiratory stable to assess for intra-thoracic causes of the dyspnoea *(figure 4 & 5)*.

Parameter	Results	Reference interval
PCV	29	30 - 50%
Total Solids	58	54 - 84/L
Glucose *(Glucometer)*	12	3.9 - 8.3 mmol/L
Plasma Lactate*	2.87	None defined for cats (0.5 - 2.5 mmol/L in dogs)

Figure 1: Venous blood gas, PCV/TS and spot glucose results. ***Plasma lactate is used as an indirect marker of anaerobic metabolism and can be a useful ancillary aid in detecting states of poor perfusion of oxygen.***

Parameter	Results	Reference interval
RBC	7.57	6.5-12.2x10*12/L
HCT	31.0	30.3-52.3%
HGB	10.4	9.8-16.2g/dL
MCV	41.0	35.9-53.1fL
MCH	13.7	11.817.3pg
MCHC	33.5	28.1-35.8g/dL
WBC	25.69	2.87-16.0x10*9/L
Neutrophils	20.91	1.48-10.29x10*9/L
Lymphocytes	3.80	0.92-6.88x10*9/L
Monocytes	0.85	0.05-0.67x10*9/L
Eosinophils	0.08	0.17-1.57x10*9/L
PLTs	6	151-600KuL

Figure 2: Haematology results. Abnormal results highlighted ***(red indicates high result, blue low result outside of reference range).***

Parameter	Results	Reference Interval
Albumin	38	22-44g/L
Globulin	39	15-57g/L
Total Protein	77	54-82g/L
BUN	11.6	3.6-10.7mmol/L
Creatinine	108	27-186ummol/L
ALT	64	20-100U/L
ALP	23	10-90U/L
Total Bilirubin	7	2-10umol/L
Glucose	6.5	3.9-8.3mmol/L
Sodium	145	142-164mmol/L
Potassium	4.8	3.7-5.8mmol/L
Calcium (Total)	2.54	2.00-2.94mmol/L
Phosphate	1.57	1.10-2.74mmol/L

Figure 3: Biochemistry results. Abnormal results highlighted ***(red indicates high result, blue low result outside of reference range).***

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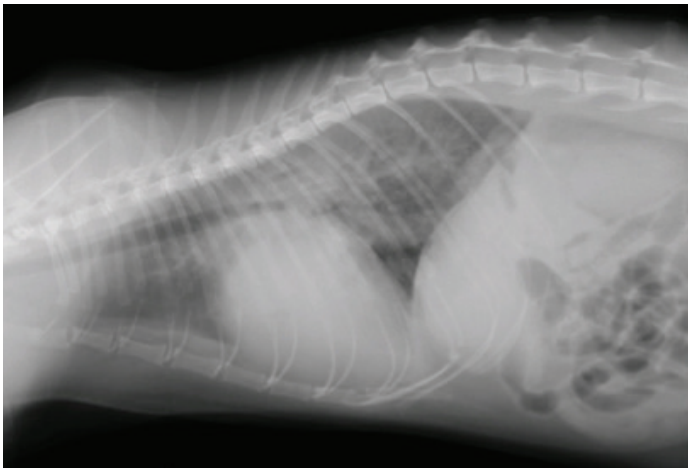

Figure 4: Left lateral thoracic radiograph (conscious).

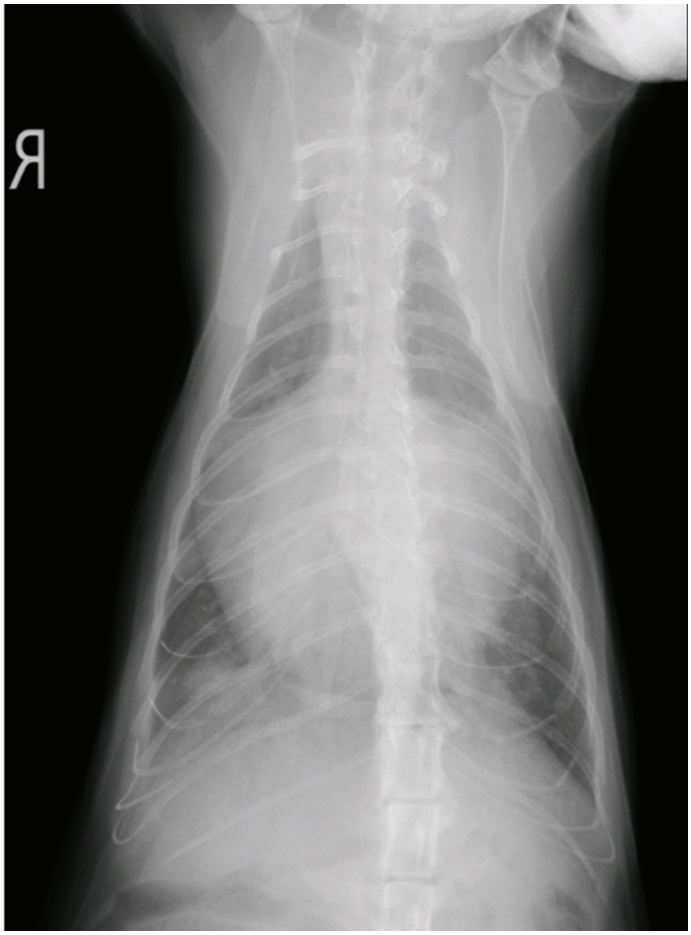

Figure 5: DV thoracic radiograph (conscious).

How would you interpret these radiographs?
There is a mixed alveolar, interstitial pattern with air- bronchograms visible caudal to the carina on the lateral view and within the left cranial lobe on the DV view. The cardiac silhouette is globally enlarged with increased sternal contact on the lateral view and increased costal contact bilaterally on the DV view. The carina is displaced dorsally. Pulmonary vein congestion is also noted on the lateral view cranial to the cardiac silhouette.

The diaphragm appears intact, and the cranial abdomen appears within normal limits.
The muscoloskeletal structures appear normal.
The radiographic findings are all suggestive of cardiac disease with congestive heart failure. This would explain the presenting clinical signs of expiratory dyspnoea, tachycardia, bilateral harsh lung sounds and pallor, as well as the jugular distension and gallop rhythm. However, this does not explain the seizure like episode, nor the severe thrombocytopenia.

What are the most likely differentials for this cat at this point?

Cardiac disease:

- Hypertrophic cardiomyopathy (HCM).
- Restrictive cardiomyopathy (RCM).
- Pericardial effusion (globoid round cardiac silhouette) although this is very rare.

Thrombocytopenia:

- Destructive (immune mediated thrombocytopenia, neoplasia, or drug reactions). -

Consumptive (haemorrhage, DIC, coagulopathy).

- Decreased production (bone marrow disease, infectious agents, drug reactions).
- Sequestration (splenomegaly, neoplasia, severe hypothermia).

Seizure like activity:

- Extracranial: metabolic or toxic less likely based on biochemistry and history, traumatic cause still cannot be excluded, hypoxic episode secondary to an embolus is a major differential combined with the thrombocytopenia.
- Intracranial: structural lesions (neoplasia, inflammatory/infectious) cannot be ruled out at this stage.
- Also consider the possibility the episode was not a seizure – other differentials could include syncope, collapse or a neuromuscular disease.

Further investigation
To further investigate the cardiac abnormalities identified on plain radiography an echocardiogram was performed to assess for structural change to the heart. Findings were consistent with a hypertrophic cardiomyopathy (HCM) with a reduced systolic function, enlarged dilated left ventricle consistent with potential left ventricular outflow tract obstruction (LVOTO), and a generalised enlarged left atrium consistent with congestive heart failure. Additionally, a large increased echogenicity was observed within the left atrium with a 'smoke' like appearance *(figure 6)*.

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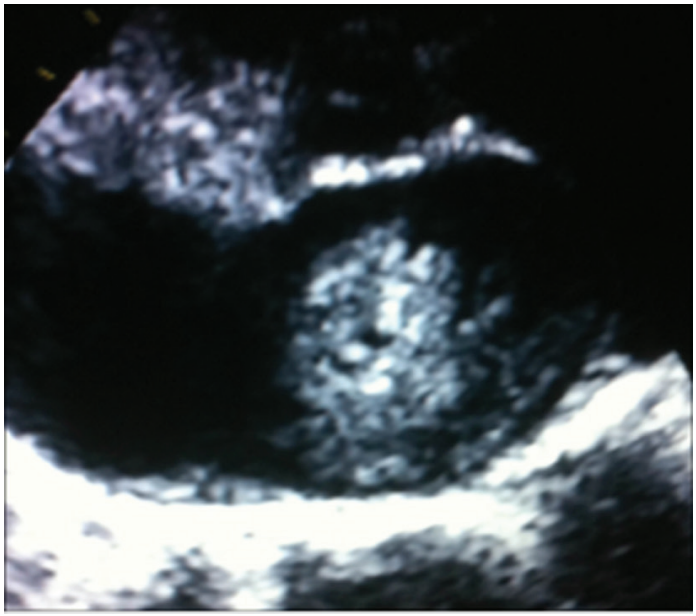

Figure 6: Dilated left atrium with a large increased echogenicity within the left atrium. Note 'smoke' like appearance in the right aspect of the atrium.

What's your final diagnosis?
HCM with congestive heart failure. The acute onset of presentation suggests this has moved into the clinical, decompensated phase of the disease. There is a large thrombus lodged within the left atrium. Consumptive thrombocytopenia is the most likely explanation for the thrombocytopenia in this case.

Case Summary and outcome:
The initial presenting signs of seizure like activity, expiratory dyspnoea with harsh bilateral lung sounds, tachycardia and pallor were suggestive, but not specific for cardiac disease; in this case the immediate stabilisation of the dyspnoea and pulmonary oedema is critical prior to attempting invasive, stress inducing procedures such as thoracic radiography. The seizure activity in this cat was presumed to be related to a vascular, embolic episode secondary to dislodgement of thrombus in the left atrium but could also be a result of hypotension/poor perfusion. No further seizure activity was observed after admission. Unfortunately, following discussion with the owners, the cat was euthanised at this point due to financial limitations and the owners being unable to tablet the cat every day in order to manage the congestive HCM.

Ten facts: HCM

- HCM is the most commonly diagnosed cardiac disease of cats.
- Cats with asymptomatic HCM can have survival times from three years, and potentially live a near-normal life span.
- Symptomatic cats have a poorer prognosis. Those with congestive heart failure have a reported median survival time (MST) of 3- 18 months, and those with aortic thromboembolism have a reported MST of just 2-6 months.
- No other risk factors for a poorer prognosis have yet been identified.
- There is no evidence to show that treatment of asymptomatic HCM improves survival time or disease progression.
- Most HCM is idiopathic.
- Feline HCM shares many characteristics with human HCM.
- As with human HCM there is thought to be a genetic element in some cats.
- Maine Coons, Ragdolls, British Shorthairs, Rex and Persian cats are at greater risk of HCM but it can and does occur in all breeds.
- Maine Coon and Ragdoll cats can be tested by Langford Diagnostic Laboratories for a gene mutation that has been linked with HCM.

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