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Cardiology Crash Course Online 'Mini Series'

Session 2:
Aquired Canine Heart Disease

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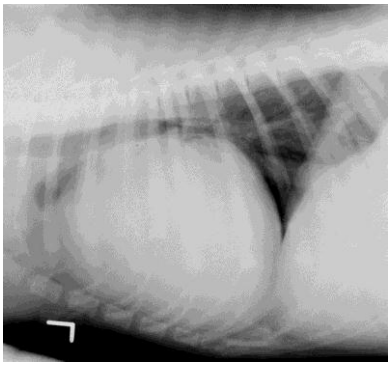

Canine Myxomatous Mitral Valve Disease

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Introduction

- Most common form of acquired canine heart disease
- Myxomatous degeneration of the mitral valve (and tricuspid valve, often to a lesser extent)
  - leading to atrioventricular valvular insufficiency
- Most common in middle to older aged dogs
- Most common in small breeds of dogs
  - Cavalier King Charles
  - Dachshund
  - Miniature Schnauzer
  - Chihuahua
- Some large breeds eg. German Shepherd
  - Develop less myxomatous thickening of MV than small breeds
  - Systolic function appears to deteriorate faster than in small breeds

Clinical presentation

- Asymptomatic
  - i.e. heart murmur detected during routine examination

or

- Typical congestive heart failure history
-

Typical congestive heart failure history

- Increased respiratory rate and effort
  - Often acute onset
- Coughing
  - Particularly at night, early morning
  - Often described as “gag” or retch
- Nocturnal restlessness

- Exercise intolerance
- Decreased appetite
- Weakness / collapse / fainting

Cardiac auscultation

- Most valuable part of cardiovascular examination
- Gives important information about heart and great vessels
- Relevant to diagnosis and treatment

Grading heart murmurs

Grade 1 - Focal and difficult to find
Grade 2 - Easily found; murmur is softer than S1 and S2
Grade 3 - Murmur is as loud as S1 and S2
Grade 4 - Murmur is louder than S1 and S2
Grade 5 - Precordial thrill is present
Grade 6 - Murmur can be auscultated with stethoscope removed from chest

Murmur of mitral regurgitation

- Point of maximal intensity is left apex (MV area)
- Radiates dorsally and to the right thorax
- Confuses identification of concurrent tricuspid regurgitation
- Murmur grade does not reliably indicate severity

Physical examination findings in asymptomatic dog with MMVD

- Murmur
  - Can be very loud!!
- Normal heart rate
- Sinus arrhythmia due to normal resting vagal tone
- Normal respiratory rate and effort
- Good peripheral perfusion
- REMEMBER THAT A LOUD HEART MURMUR DOES NOT MEAN THAT THE DOG IS IN CHF!
- IF SINUS ARRHYTHMIA IS PRESENT, THE DOG IS NOT IN CHF!

Physical examination findings in dog with CHF secondary to MMVD

- Murmur
  - usually at least a Grade 4-6/6
- Tachycardia
  - sinus tachycardia with loss of sinus arrhythmia
- Arrhythmias
  - APCs, atrial fibrillation most common
- Pulmonary crackles
  - Cardiogenic pulmonary oedema
- Tachypnoea / dyspnea
- Signs of poor peripheral perfusion
- +/- ascites and jugular distension (right-sided CHF)
- +/- Weight loss / cachexia (advanced CHF)

Pulmonary hypertension in MMVD

- Chronic elevation of LA pressure causes increase in pulmonary venous pressure, and results in pulmonary capillary hypertension
- PH resulting from MMVD (and DCM) is typically in the mild-moderate range (rarely severe)
- Variable clinical symptoms in dogs with PH:
  - None
  - May have right-sided CHF (typically ascites)
  - May have dyspnoea
    - Unrelated to cardiogenic pulmonary oedema
  - May have exercise intolerance, syncope on exertion and exercise

Initial diagnostic work-up

- Radiographs
  - Cardiac enlargement
  - <u>ESSENTIAL</u> for diagnosis of CHF
  - Left sided-CHF
  - Pulmonary oedema
  - Pulmonary vein congestion

Further diagnostic tests

- Electrocardiography
  - +/- tachycardia
    - sympathetic stimulation with CHF
  - +/- arrhythmia
    - APCs, atrial fibrillation
    - VPCs less common

- Haematology
  - Typically normal

- Biochemistry
  - May see pre-renal azotaemia in CHF
  - Otherwise non-specific, but important as baseline for medication monitoring
  - Renal parameters and electrolytes with ACE inhibitor and diuretic therapy

- Cardiac biomarkers
  - Still uncertainty about their role in a practice setting
  - NT-pro BNP
  - Cardiac troponin I (cTnI)

- Echocardiography
  - Ultrasound is the only way to definitively diagnose type and extent of underlying heart disease
  - Provides in-depth analysis of cardiac function
  - Does NOT diagnose pulmonary oedema!

Natural history of disease

- Progression of compensated heart disease to heart failure happens over many years
- Key question that we need to answer in each individual patient with MMVD is where they are along that time course of progression.
- This is vital information when making decisions concerning treatment, follow-up and prognosis

Prognosis

- Variable depending on how far advanced the disease is at diagnosis
- Dogs can have murmurs for many years before developing CHF
- If develop the disease late in life, may die of other non-cardiac cause
- Once in CHF, life expectancy is several months to several years (the latter being less common)

Canine Dilated Cardiomyopathy

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Introduction

- Primary myocardial disease
- The second most common acquired cardiovascular disease in dogs
- Typically effects large and giant breeds of dog
- Usually effects pure-breeds:
  - Dobermanns
  - Irish Wolfhounds
  - Great Danes
  - Newfoundlands
  - Boxers (ARVD)
  - Cocker Spaniels
- Affects all ages
  - Increased risk with increased age
  - Juvenile onset form seen in Portugese Water Dogs (2-32 weeks old)
- Possible male predisposition in *some* breeds?
- Males may be effected earlier and progress faster?

Proposed causes:

- Genetic
  - Most likely cause in our patient population

- Nutritional deficiencies (rare)
  - E.g. taurine
- Myocardial toxins (uncommon)
  - E.g. doxorubicin
- Persistent or recurrent tachycardia (uncommon)

Declining myocardial contractility

- Impaired systolic function
- Decreased rate of ventricular pressure development
- Reduced ventricular ejection
- Increased end-systolic volume
- Decreased myocardial compliance
- Impaired diastolic function

The following also contribute to ventricular dysfunction

- Arrhythmias
  - supraventricular (AF)
  - ventricular (VT)
- AV valvular insufficiency
  - mitral and tricuspid regurgitation, usually secondary to stretching of AV valve annulus

Clinical presentation in breeds such as Dobermanns

- Acute LEFT sided congestive heart failure
  - Coughing (often mistaken for kennel cough)
  - Dyspnoea

- Life threatening ventricular arrhythmias
  - syncope
  - episodic weakness
  - sudden death
  - 20-30% Dobes and Boxers with DCM die suddenly before onset of CHF

Clinical presentation in other breeds, such as Irish Wolfhounds, Great Danes, Newfoundlands, Cocker Spaniels

- Syncope and sudden death less likely
  - Ventricular arrhythmias and sudden death in UK Great Danes with DCM is more common than previously thought
- Ascites/pleural effusion (RIGHT sided CHF signs) more likely
- May have more subtle signs eg exercise intolerance
- May be detected on discovery of heart murmur or arrhythmia (e.g. Atrial fibrillation)

Boxer cardiomyopathy / ARVC

- Inherited form of myocardial disease
- Characterised by ventricular tachycardias, syncope, +/- ventricular systolic dysfunction and CHF
- Seems to be similar to Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) in humans
  - Fatty infiltration of myocardium (especially RV) on histopathology
- Variable prognosis
  - Sudden death
  - Death from CHF
  - Some dogs can be managed on antiarrhythmics (+/- intermittent collapse) for years

Physical examination

- Approx 50% dogs with DCM have soft regurgitant systolic murmur of MR
  - Grade I-III/VI
  - Audible over MV and/or TV area
  - MR develops due to stretching of MV annulus as LV chamber dilates

- Irregular heart rhythm
  - Atrial fibrillation
  - Atrial premature complexes (APCs), runs of SVT
  - Ventricular premature complexes (VPCs), runs of VT

- S3 gallop sound
- Diastolic dysfunction
- Weak, rapid pulses
- Pulse deficits
- Pulmonary crackles
- Dull lungs, heart – pleural effusion
- Jugular venous distension, pulsing
- Hepatomegaly
- Ascites
- Weight loss/muscle wasting

ECG abnormalities

- Wide, tall QRS – left ventricular enlargement
- Wide P waves – left atrial enlargement
- NOT present in all affected dogs
- Arrhythmias – breed differences
  - Dobermanns, Boxers
    - very high prevalence of VT (>80%)
    - low prevalence of AF (<30%)
    - 24 hour Holter monitor recording indicated to properly evaluate arrhythmias and decide on best treatment options

Radiographic findings

- Giant and large breeds and Cockers
  - generalised cardiomegaly
  - Biventricular CHF
- Dobermanns, Boxers
  - Can be less impressive cardiomegaly
  - LA enlargement / pulmonary oedema

- Heart size does not correlate with clinical outcome

Echocardiography

- To make definitive diagnosis of DCM

- To define extent of systolic and diastolic dysfunction
- Hallmarks of DCM on echo:
  - Chamber dilation
  - Reduced myocardial contractility
  - Fractional shortening usually is < 10-15% (normal = 25-40%)

Other diagnostic tests

- Haematology
  - Typically normal

- Biochemistry
  - May see pre-renal azotaemia in CHF
  - Otherwise non-specific, but important as baseline for medication monitoring
  - Renal parameters and electrolytes with ACE inhibitor and diuretic therapy

- Cardiac biomarkers
  - Still uncertainty about their role in a practice setting
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Occult DCM

- Most work done in Dobermanns
- Slowly progressive insidious disease
- Protracted subclinical, “occult” phase
- Can last several years prior to onset of CHF
- Characterised by ventricular arrhythmias and left ventricular dysfunction
- <u>N.B. Dobermann with a normal echo and no VPCs can develop DCM at ANY time subsequently</u>

Prognosis

- Always terminal, unless underlying cause can be identified and reversed
- Variable survival times, despite optimal therapy
- Disease progresses at different rates in different breeds
- Rapid progression in Dobermanns and Great Danes

- Other breeds have slower progression
  - E.g. Cockers, Newfoundlands

Infective Endocarditis

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Introduction

- Infective endocarditis (IE) is a disease associated with high morbidity and mortality

Definition

- IE occurs when there is microbial invasion into endothelium of heart valves or endocardium
- In dogs, aortic valve and mitral valve are most commonly affected
- Reported prevalence is low, but may be underestimated as this condition has non-specific clinical signs and is difficult to diagnose

Predisposing Factors

- Requires bacteraemia and endothelial disruption
- Sub-aortic stenosis (SAS) is the most common predisposing cardiac defect in dog
- Myxomatous mitral valve disease (MMVD) does not seem to be a predisposing factor for IE
- Common sources of bacteraemia include discospondylitis, prostatitis, pneumonia, UTI, pyoderma, periodontal disease, long term indwelling central venous catheters

Pathogenesis

- Lesions form on ventricular side of aortic valve and atrial side of mitral valve
- Once clot forms on damaged endothelium, bacteria can bind (bacteraemia)
- Fibrinous vegetative lesion shields bacteria from blood stream and host defenses
- Hard for antibiotics to penetrate
- Most common: - Staph spp. (aureus, intermedius, coag +ve and -ve); Strep spp. (canis, bovis, B-haemolytic); E.Coli
- Less common include: - Pseudomonas, Enterobacter, Pasteurella
- Bartonella is most common cause of culture- negative IE in dogs
- IE bacteria secrete enzymes that destroy valve tissue and rupture CT
- Platelets release bactericidal proteins but most bacteria that cause IE are resistant to these proteins
- Staph and Bartonella can become internalised within the endothelial cells and therefore escape detection

by immune system

Bartonella IE in dogs

- Most commonly affects aortic valve
- Unique valvular lesion characterised by fibrosis, mineralisation, endothelial proliferation, neovascularisation
- Colonises RBCs and endothelial cells
- Impairs immune system

Risk Factors for IE

- Unclear whether immuno-suppression plays a role in development of IE
- Dental prophylaxis does not seem to be a predisposing factor in dogs (Reference: Peddle GD. JAVMA 2009)
- American Heart Association (AHA) revised guidelines on prevention of IE in humans, published in Circulation 2007: guidelines exclude dental prophylaxis as a procedure requiring peri-operative antibiotics

Pathogenesis

- Dogs with IE develop high titres of antibodies against causative microorganism
- Leads to continuous formation of circulating immune complex
  - Polyarthritis, glomerulonephritis
- Septic and non-septic thromboembolism (especially with mitral valve IE)
  - Infarction of kidneys, spleen, myocardium, brain

History

- Often ill-defined history with non- specific signs:
  - Lethargy, weakness, weight loss, lameness, anorexia, fever (can be episodic)

Signalment

- Typically large breed dogs (>15 kg)
- Middle to older age
- Male

Clinical signs

- Heart murmur
  - Systolic murmur of mitral regurgitation with mitral valve IE
  - Diastolic murmur of “new” aortic regurgitation caused by aortic valve IE and pre-existing systolic murmur of aortic stenosis (predisposing factor), can cause characteristic “to and fro” murmur
- Bounding pulses (if aortic valve IE, due to increased pulse pressure difference with aortic regurgitation)

Clinical signs

- Tachycardia, pallor
- Fever
- Lameness
- Joint pain, swelling
- Neurological abnormalities
- NOT exhaustive list!

Diagnosis

- Bloods
  - Leucocytosis, mature neutrophilia and monocytosis
  - Thrombocytopaenia, anaemia
  - Increased D-dimer and FDPs
  - Hypoalbuminaemia, raised liver enzymes, renal failure, acidosis

- Urine
  - Cystitis, proteinuria, haematuria – UPC – Urine culture

- Joint fluid analysis and culture

- Blood culture
  - Aseptically collected 30 - 60 mins apart
  - Need decent volume of blood as bacterial concentration is low
  - Submit for aerobic and anaerobic culture
  - High probability of negative blood culture if dog already on antibiotics (take blood culture during trough of antibiotic level in blood)

  - N.B. Bartonella is intracellular bacterium so difficult to culture from blood or tissues
    - Therefore diagnosis is limited to PCR of blood or PCR of infected valve at post mortem

- Thoracic radiography
  - Pulmonary oedema and pulmonary venous congestion without cardiomegaly (due to acute nature of CHF)

- ECG
  - Arrhythmias including ventricular and supraventricular tachycardias and third degree AV block

- Echocardiography
  - Most important diagnostic tool
  - Hyperechoic oscillating, mobile, irregular shaped mass adherent to, yet distinct from, the endothelial cardiac surface
  - IE causes moderate to severe valvular insufficiency
  - Differential diagnosis on echo is MMVD, but signalment is different!
    - i.e. MMVD typically seen in small breeds vs. IE which is typically seen in large breeds
    - Large breeds with age-related MMVD do not develop such marked valvular thickening as is seen with valvular IE

Suggested diagnostic criteria for IE in dogs

- Major Criteria:
  - Vegetative lesion on valve/endocardium on echo
  - New valvular insufficiency • Positive blood cultures
  - 2 or more +ve blood cultures OR 3 or more with common skin contaminants

- Minor Criteria:
  - Fever
  - Medium to large breed dog (>15 kg)
  - SAS
  - Thromboembolic disease
  - Immune mediated disease
  - Positive blood culture not meeting major criteria
  - Bartonella serology consistent with infection

How to use diagnostic criteria for IE:

- Definite
  - Pathology of valve
  - 2 major criteria
  - 1 major and 2 minor criteria
- Possible
  - 1 major and 1 minor
  - 3 minor
- Rejected
  - Firm alternative diagnosis
  - Resolution in < 4 days treatment
  - No pathological evidence
  -

Treatment

- Treat long term (8-12 weeks) broad spectrum antibiotics, ideally including a minimum of 1 week of IV antibiotics
- Start empirical antibiotic treatment whilst cultures pending
- Antibiotics must have good tissue and intracellular penetration

Treatment and Follow-up

- Echo (repeat 1-2wks after starting antibiotics; repeat 4-6 wks into course; repeat 2 weeks after stopping antibiotics)
- Temp, CBC, urine serially
- Blood culture (repeat 1 week after starting antibiotics and 2 weeks after stopping antibiotics)
- Bartonella - doxycycline, fluoroquinolones, azithromycin

Antibiotic Prophylaxis

- It is recommended to give peri- operative antibiotics in dogs with SAS
  - E.g cephalosporin – Give 1 hour before surgery or dentistry and 6 hours after
- In MMVD, peri-operative antibiotics are not currently a recommendation

Prognosis

- Depends on which valve is infected
  - Aortic valve - grave prognosis (aortic regurgitation is poorly tolerated by LV)
  - Mitral valve - poor to fair prognosis (mitral regurgitation is better tolerated by LV)

Pericardial Disease

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Introduction

Pericardial diseases comprise a small proportion of clinically important cardiovascular disease in dogs and cats. Pericardial disease is one of most important causes for right-sided congestive heart failure in dogs. Therefore it is important to distinguish it from valvular and myocardial disease. Pericardial disease is rare in cats.
The clinical signs can be subtle, although the consequences can be life-threatening.

Anatomy of the pericardium

- Heart is enveloped in the pericardial sac
- Composed of 2 layers:
  - Tough outer fibro-serous membrane (parietal pericardium)
  - Delicate inner serous membrane (visceral pericardium) = epicardium
- Pericardial cavity is formed between the 2 layers
- Normal dogs and cats have approx. 0.25 ml/kg pericardial fluid in pericardial cavity

Mechanical properties of the pericardium

- Very distensible when first filled
- Non-distensible when full
- Adaptive response by pericardium to chronic increases in volume = hypertrophy (growth)
- This serves to increase reserve volume and compliance

Definition of cardiac tamponade

"An impairment of ventricular filling as a consequence of increased intrapericardial pressure caused by the accumulation of fluid within the pericardial cavity"

Pathophysiology of cardiac tamponade

- As pericardial fluid accumulates, intrapericardial pressure rises
- More pressure is therefore needed to push blood into the right and left ventricles during diastole to maintain cardiac output
- As the intraperiardial pressure continues to increase, cardiac output decreases and systemic venous pressure increases

- The severity of circulatory compromise is a function of intrapericardial pressure rather than volume of pericardial fluid
- When intrapericardial pressure rises **gradually**, typically see signs of **systemic congestion** eg. ascites, pleural effusion
  - Systemic capillaries leak badly at pressures between 10-15 mmHg
  - Pulmonary capillaries do not leak significantly until pressures reach 30 mmHg
  - This is why patients with chronic cardiac tamponade present with right-sided congestive heart failure signs (typically ascites) rather than left-sided congestive heart failure signs

- But if intrapericardial fluid increases **abruptly**, may result in **cardiogenic shock** before signs of systemic congestion are evident
  - Usually caused by trauma and bleeding into pericardial sac

Pericardial diseases of dogs

- Neoplasia (most common)
  - Hemangiosarcoma (typically on right atrium)
  - Chemodectoma
  - Mesothelioma

- Idiopathic Pericarditis

- Other
  - Left atrial rupture
  - Coagulopathy
  - Infectious

Pericardial effusions in dogs

- Most diseases that effect the pericardium can result in development of pericardial effusion and cardiac tamponade
- Only a few frequent causes:
- > 90% of pericardial effusions in dogs are idiopathic or neoplastic
- Majority of effusions in dogs are sanguinous (haemorrhagic), regardless of whether idiopathic or neoplastic

Pericardial effusions in cats

- Less common in cats than in dogs
- Most common causes:
  - FIP
  - CHF
  - Infection
  - Neoplasia (e.g. lymphosarcoma)

Idiopathic pericardial effusions in dogs

- Inflammatory condition
- Large, giant breed, male, middle age
  - E.g. St Bernard, Golden Retriever
- Inflammation of the blood vessels and lymphatics within the pericardium = source of haemorrhagic effusion
- Slow to accumulate
- Self limiting or may recur (months to years)

Neoplastic pericardial effusions in dogs

- Haemangiosarcoma
  - Originate from right atrium/right atrial appendage
  - E.g. German Shepherd
  - Acute or chronic tamponade
  - May be significant metastasis
  - Lungs, liver, spleen, kidney
  - Arrhythmias, systemic illness

- Chemoreceptor cell tumours (Chemodectoma)
  - Develop around root of aorta
  - Metastasis is rare
  - Space occupying
  - Can cause arrhythmias
  - Brachycephalic breeds, male
  - Usually clinical signs only develop when pericardial effusion present

- Mesothelioma
  - Rare tumour arising from serous membranes
  - Tends to metastasize
  - Haemorrhagic effusions without distinct mass lesion on echo
  - Pericardium may appear thickened/nodular (not always)
  - Definitive diagnosis by exploratory thoracotomy and biopsy

Other causes of pericardial effusion

- Pericardial infection
  - Uncommon in dogs (more common in cats, although still rare)
  - Purulent effusion with inflammatory cytology, +/- bacteria/fungi
- LA rupture
  - Dogs with advanced MMVD and severe mitral regurgitation
  - Small breed dogs

Clinical signs of cardiac tamponade

- Clinical manifestations depend on RATE of rise of intrapericardial pressure
- Most animals present with chronic history (less severe signs)

- Acute pericardial effusions are less common
  - Signs include collapse, shock, dyspnoea, sudden death
  - E.g. atrial tear, bleeding tumour, traumatic laceration of coronary vessels

- With a chronic effusion, there is usually a vague history of lethargy, weakness, exercise intolerance, anorexia, weight loss
- 3 typical clinical findings:
  - Muffled heart sounds
  - Weak arterial pulses
  - Distended jugular veins
- Signs of right-sided congestive heart failure may be present i.e. ascites or pleural effusion

Thoracic radiography

- Globular, round appearance to cardiac silhouette
- Well defined borders of cardiac silhouette as heart is beating within relatively static fluid filled sac
- Distension of caudal vena cava, hepatomegaly and ascites may be seen with chronic effusions (right-sided CHF).
- Enlargement of the pulmonary veins and pulmonary oedema (left-sided CHF) is a rare finding

Electrocardiography (ECG)

- Sinus tachycardia and small amplitude QRS complexes are recorded most frequently in dogs and cats with substantial pericardial effusion
  - Non-specific finding (esp. in cats)
- Electrical alternans may be present – beat to beat variation in amplitude of QRS complexes as heart swings within fluid filled pericardial sac
  - NOT present in all cases of pericardial effusion

Echocardiography

- Safest, most practical and most cost-effective method for diagnosing pericardial effusion
- Pericardial fluid is represented by an anechoic space between the epicardium and pericardium

- Allows estimation of volume of pericardial effusion (> volume more likely to be significant)
- N.B. pressure within the pericardial sac is more important than volume
- Diastolic collapse of right atrium/right ventricle is good evidence of increased intrapericardial pressure

- Echo cannot provide histologic diagnosis of a mass if present
- But location and appearance of many mass lesions are highly suggestive of their identity
- E.g. In dogs, a right atrial mass is most likely a haemangiosarcoma, and a mass located at the root of the aorta is most likely to be a chemodectoma

Pericardiocentesis

- Pericardiocentesis is **vital** if tamponade is present!

- **If sedation is necessary for pericardiocentesis, stabilise cardiovascularly compromised patient in cardiac tamponade with IV fluid therapy NOT DIURETICS!!!**
  - **Diuretics will decrease vascular volume and BP and will not decrease fluid accumulation within pericardial sac**

- Dramatic clinical improvement after drainage
- Improved demeanour, decrease in HR, improved pulse quality, resolution of ascites and jugular distension
- Resolution of electrical alternans, increased QRS amplitude

Pericardial fluid analysis

- Diagnostic potential of cytology is limited for typical haemorrhagic effusions
  - Many intrapericardial neoplasms do not readily exfoliate
  - High number of reactive mesothelial cells occur in all pericardial fluid samples
- pH and cardiac troponin (cTnI) have not proved reliable ways to distinguish between idiopathic and neoplastic effusions in dogs

Treatment and prognosis of Idiopathic effusions

- Initial pericardiocentesis
- Effusion may or may not recur (approx 50% recur)
- Steroid use is not validated in dogs
- If recurs, requiring drainage > 3 times, typically recommend thoracotomy and pericardectomy
- ALWAYS submit pericardium for histopathology to rule out mesothelioma
- **Good long-term prognosis** if no neoplastic aetiology present

Treatment and prognosis for neoplastic effusions

- Initial pericardiocentesis
- Effusions will recur within days/weeks
- Require repeat drainage (palliative)
- Prognosis very guarded, esp. for haemangiosarcoma, and chemotherapy protocols are of limited benefit
- Thoracotomy and pericardectomy for biopsy/debulking (confirmation of diagnosis) – unlikely to improve prognosis with haemangiosarcoma, but may give symptom-free months to years with chemodectoma