“Pericardial Effusions – Diagnosis and Treatment”

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PERICARDIAL DISEASE - DIAGNOSIS AND TREATMENT

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Estimated to make up around 7% of canine heart disease, less common in the cat.

A) **ANATOMY** - The parietal pericardium consists of an outer fibrous layer and an inner serosal layer. The visceral pericardium, or epicardium, is a reflection of the serosal surface of the parietal pericardium. The pericardial cavity between the parietal and visceral pericardia is a potential space. In healthy individuals, it contains a small volume of serous transudate. The normal pericardium appears to fulfill only a minor role in cardiovascular function and its congenital absence is clinically silent. However, disease of the pericardium impairs cardiac filling and this can lead to marked cardiovascular compromise. Congenital abnormalities of the pericardium are relatively uncommon. During this session, the emphasis will be on acquired pericardial disease which occurs in essentially two forms. Effusive pericardial disease is most common; it is defined by the accumulation of an abnormally great volume of fluid within the pericardial space. Constrictive pericardial disease is uncommon in all veterinary species. Pericardial constriction usually results from fibrosis of the parietal or visceral pericardium, which limits ventricular expansion and therefore impairs ventricular filling. Pericardial disease is observed in both dogs and cats but is seldom the cause of clinical signs in the latter. This review will primarily address the diagnosis and therapy of pericardial effusion in the dog.
B) **PERICARDIAL EFFUSION**

**AETIOLOGY:**
In dogs, pericardial effusion (PE) usually is hemorrhagic; numerous causes have been reported but most commonly, canine PE is due to intrapericardial neoplasia or idiopathic pericarditis. Right atrial hemangiosarcoma (HSA) and chemodectoma are the most common cardiac neoplasms but mesothelioma, ectopic thyroid carcinoma, rhabdomyosarcoma and metastatic neoplasms occasionally cause PE. Except in specific geographical regions, infective pericardial disease is rare.

<table>
<thead>
<tr>
<th>Type of Fluid</th>
<th>Cause</th>
<th>Diagnostic Features</th>
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</thead>
<tbody>
<tr>
<td><strong>HAEMORRHAGIC</strong></td>
<td>Idiopathic: could it be immune-mediated??</td>
<td>Defibrinated blood/sero-sanguineous, sterile. Pericardial fluid may be acid pH (unreliable)</td>
</tr>
<tr>
<td></td>
<td>Heart base Tumours</td>
<td>Older, &gt;8 years, brachycephalics. Uncommon</td>
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<td></td>
<td>Haemangiosarcoma: commonest in G. Shepherd. Probably the commonest aetiology</td>
<td>Right auricle is commonest location: may occur as acute tamponade/death, or as more gradual accumulation &amp; RSCHF. Bloody fluid, may be alkaline pH (unreliable)</td>
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<tr>
<td></td>
<td>Left atrial rupture</td>
<td>Uncommon, 2°C to Mitral insuffic. jet lesions, occurs as acute tamponade, often fatally</td>
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<tr>
<td><strong>TRANSUDATE</strong></td>
<td>RSCHF</td>
<td>Often present, but clinically not detectable</td>
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<tr>
<td></td>
<td>Hypoproteinaemia</td>
<td>Uncommon</td>
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<tr>
<td><strong>EXUDATE</strong></td>
<td>Penetrating wound</td>
<td>Uncommon</td>
</tr>
<tr>
<td></td>
<td>Haematogenous</td>
<td>Various agents, uncommon</td>
</tr>
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**PATHOPHYSIOLOGY:**
As the volume of pericardial fluid increases, the pressure begins to affect atrial and ventricular filling, particularly the lower pressure right side, thus producing signs predominantly of right-sided congestive heart failure, and progressive cachexia. The reduced end-diastolic volume results in signs of forward heart failure and tachycardia. If fluid in the sac accumulates slowly, a remarkably large volume can be present. Our present "record" for idiopathic haemorrhagic effusion is 3 litres!

The consequences of pericardial effusion (PE) depend on the volume of pericardial fluid and on the rate at which it accumulates. Acutely, the pericardium is minimally distensible. Therefore, when PE accumulates rapidly, relatively small volumes cause intrapericardial pressures (IPP) to rise resulting in impaired ventricular filling and hemodynamic compromise. In contrast, the pericardium can stretch to accommodate an effusion that develops slowly and large volumes of fluid may accumulate before IPP impairs cardiac filling.
The syndrome of cardiac compression that results from accumulation of pericardial fluid is known as cardiac tamponade. Right atrial and ventricular pressures normally are lower than corresponding pressures of the left atrium and ventricle; because of this, the right side of the heart is affected initially by tamponade. Progressive increases in intrapericardial pressure cause equalization of left and right ventricular filling pressures after which further increases in IPP cause ventricular filling pressures to rise in tandem. The increase in ventricular filling pressures causes venous pressures to increase, and when PE is chronic, signs of systemic congestion including ascites and pleural effusion are observed. Severe tamponade reduces stroke volume and results in systemic hypotension. When pericardial fluid accumulates rapidly, clinical signs of diminished peripheral perfusion usually dominate the clinical presentation. Signs of systemic congestion generally develop at venous pressures that are lower than those that cause pulmonary congestion. Most veterinary patients with PE are therefore presented for evaluation of signs such as ascites. Pulmonary edema is uncommon in the setting of tamponade.

**Pulsus Paradoxus:**
Pericardial effusion and tamponade enhance ventricular interdependence potentially resulting in pulsus paradoxus. Pulsus paradoxus is an exaggeration of physiologic changes in cardiac loading that normally result from respiratory-associated variations in intrathoracic pressure. With respect to acute forces, the fibrous pericardium can be considered inelastic and therefore, the total volume of the pericardium does not vary in the short-term. During inspiration, intrathoracic pressure falls and this augments right ventricular filling. Because the total volume of the pericardium is fixed, the increase in right ventricular volume occurs at the expense of left ventricular filling and stroke volume. The result is a decrease in the strength of the arterial pulse during inspiration.

**CLINICAL EXAMINATION:**
Muffled/diminished heart sounds, tachycardia. If chronic similar to right-sided CHF: ascites, venous congestion, dyspnoea due to pleural effusion, diminished peripheral pulse pressure & possibly pulsus paradoxicus. If effusion is acute and severe patients may present recumbent in circulatory collapse.

**ECG:**
May be diminished amplitude QRS- not reliable. Occasionally electrical alternans (varying R wave amplitudes due to “moving” heart oscillations within the sac).

**RADIOLOGY:**
Larger volumes produce classical globoid "soccer ball" heart shadow which "flattens" against chest wall and diaphragm. Pleural fluid may obscure the details of the heart. Large caudal vena cava and ascites are often noted. Fluoroscopy reveals lack of normal heart movement.

**ECHOCARDIOGRAPHY:**
Diagnostic test of choice. Difficult to assess thickness of the pericardium. Note the tendency of the RA to collapse under the pericardial pressure. Look carefully for masses near the atria and heart base, which are best seen before fluid is removed. However, right atrial haemangiosarcomas may be too small to easily identify.
MANAGEMENT:
Drain fluid - right-sided approach. Use large-bore catheter with side holes, under local anaesthetic & sedation if required. Drain as completely as possible. Fluid may appear very bloody, but is very dark and does not clot (to distinguish from fresh blood). However, sometimes haemangiosarcomas start to bleed during drainage: so stop if the bloody fluid starts to clot, and consider transfusing!
Analyse fluid: tumour cells are seldom found. A study has suggested that pH of fluid may be helpful: pH < 7.0 = benign haemorrhagic effusion, ph > 7.5 = pericardial tumour. Accuracy of this is poor!
~50% of benign idiopathic effusions resolved by a single drainage. If fluid recurs, recommend partial pericardectomy. Repeated drainage leads to pericardial fibrosis. Pericardectomy will resolve many of the recurring cases. Pericardectomy via thoracoscopy can be performed.
Some heart base tumours and right atrial haemangiosarcomas are removable. Chemotherapy (eg Doxycycline) has been applied: value yet to be proven. Partial pericardectomy may relieve signs for a while.

MAJOR DIFFERENTIAL:
The primary differential for a globoid heart on radiographs is dilated cardiomyopathy (DCM) or other underlying cardiac disease. Key findings to try to differentiate DCM from pericardial effusion without echocardiography include:

- **Heart sounds**: Heart sounds in dogs with DCM are frequently normal in contrast to the decreased heart sounds seen in pericardial effusion. A systolic murmur may be noted in dogs with DCM and is uncommon in dogs with pericardial effusion.

- **ECG**: Atrial fibrillation is not uncommon in dogs with DCM. Atrial fibrillation is uncommon in dogs with pericardial effusion. Electrical alternans may be seen in dogs with pericardial effusion.

- **Cardiac Silhouette**: The silhouette of the heart on thoracic radiographs of dogs with pericardial effusion tends to be extremely round with sharp borders. The silhouette of the heart in dogs with cardiomyopathy can be round, but often, there are still some dimples or "waist" associated with the divisions between the chambers and the borders of the cardiac silhouette tend not to be as sharp because of motion artifact.

- **Pulmonary infiltrate**: Pulmonary edema is common in DCM and uncommon in pericardial effusion.

- **Pulsus paradoxus**: Common in pericardial effusion, uncommon in DCM.
** NOT GIVE FRUSEMIDE TO A SUSPECTED PERICARDIAL EFFUSION:**

Diuretic agents are unlikely to mobilize the effusion but are likely to decrease venous pressures and cardiac output. For this reason, diuretics are contraindicated in the setting of tamponade. If pericardiocentesis must be delayed, intravenous fluid infusion is the appropriate supportive treatment.

PERICARDIOCENTESIS
You will need:
- Lidocaine 0.5cc-1.5cc
- 25-60 cc syringe
- 3-way stopcock
- 2 IV extension sets with IV catheter (24-14 gauge, long) or butterfly catheter (25-19g)
- Two plain tubes--labels with patient name and source site
- One EDTA tube labeled with patient name and source site
- Collection bowl and/or measuring device
- Sedation or anesthesia induction agent, analgesia
- Narcotic/ benzodiazepine IV, IM
- Propofol IV
- Clip/prep body cavity
- IVC/ IVF
- Flow by oxygen

Patients are on ECG monitoring, IV catheter in place, IV fluids, narcotic sedation +/- benzodiazepine and all supplies available. Patient are usually placed in left lateral recumbence.

**Step-by-step Ultrasound procedure:**
1. Clip right side of patient between the 4-6 intercostal space
2. Evaluate site with ultrasound
3. Surgically scrub area
4. Lidocaine infusion at centesis site
5. Insert a 14 gauge 5 inch long fenestrated catheter at the point of maximal effusion between the pericardial wall and heart with ultrasound guidance
6. Once see catheter enter pericardium advance 5 more millimeters and remove needle while advancing the catheter into the pericardial sac. *** Watch closely for ventricular arrhythmias, especially during this step!!!
7. Catheter then connected to extension set attached to a 3 way stopcock
8. 35-60 cc syringe attached to stopcock
9. Mild suction applied and fluid removed until negative suction is obtained, this may take a small amount of manipulation of the catheter
10. Retain a sample for cytology and culture; monitor for clotting
11. It is common that these patients also have pleural effusion present. Once the pericardial effusion is removed, remove the catheter from the pericardium and remove the thoracic effusion. Save and submit this fluid for analysis

To determine the site of pericardiocentesis it is easiest to look with ultrasound prior to
centesis. When ultrasound is not available, a radiograph can be used to help guide centesis site. If the animal is not stable enough and needs centesis immediately then perform centesis on the right side between rib space 4-6 just dorsal to the costochondral junction. The right side of the chest is used due to the cardiac notch where lungs are not as likely to get damaged. Additionally, the right side does not have the coronary vessels present so again they are not likely to be damaged.

Blind centesis can be performed in situations where ultrasound is not available. Radiographs can aide in determining a centesis site. Based on the radiograph you can estimate the intercostal space and how far in the heart is for insertion estimate depth.

**Step-by-step blind procedure:**
1. Clip right side of patient between the 4-6 intercostal space
2. Surgically scrub this area
3. Lidocaine infusion at centesis site just dorsal to the costochondral junction
4. Insert a 14 gauge fenestrated 5 inch long
5. Catheter then connected to extension set attached to a 3 way stopcock
6. 35-60 cc syringe attached to stopcock
7. Advance once the catheter is in the chest and apply mild suction
8. Once the catheter contacts the pericardium you will feel a scratching sensation, at that point only slight advancement is needed to penetrate the pericardium.
9. Once the catheter/ needle enters the pericardium, back out the needle while advancing the catheter
10. Mild suction applied and fluid removed until negative suction is obtained, this may take a small amount of manipulation of the catheter
11. Retain a sample for cytology and culture
12. It is common that these patients also have pleural effusion present. Once the pericardial effusion is removed remove the catheter from the pericardium and remove the thoracic effusion. Save and submit this fluid for analysis.

**Prognosis:**
The prognosis associated with effusive pericardial disease is largely determined by the etiology of the effusion. In general, patients with cardiac HSA have a poor prognosis. The prognosis after pericardiectomy for patients with chemodectoma may be surprisingly good presumably because the tumors grow relatively slowly and are late to metastasize. Although prolonged survival has been documented, patients with pericardial mesothelioma generally fare poorly and have decreased survival relative to dogs with idiopathic PE. Patients with idiopathic pericardial effusion have a good or excellent prognosis. A few clinical findings provide prognostically useful information. Patients that have ascites when first presented for veterinary evaluation live longer than those that do not. Presumably this is because patients with aggressive, hemorrhagic tumors such as HSA are more apt to develop signs associated with circulatory collapse than signs of congestion. Independent of histologic findings, the prognosis is generally poor for patients with an echocardiographically identified mass that originates from the right atrium.
C) CONSTRUCTIVE PERICARDIAL DISEASE
Rare in dogs and cats - result of previous trauma or infection, or from repeated previous drainage. Effect is similar to pericardial effusion. Surgical removal of pericardium has limited success, due to concurrent epicardial thickening.

D) CONGENITAL PERITONEAL/PERICARDIAL HERNIA
Rare, more cases reported in Weimeraner dogs and in cats. May be minimally symptomatic unless there is significant organ entrapment. Signs may be related to cardiac compression, or may be vague GI signs due to bowel blockage. May hear bowel signs over heart, or heart may be muffled. Often not diagnosed until well into adulthood. Most cases easily confirmed with radiology (+/- G/I contrast) or ultrasound. Treatment not always required, but surgical repair is not too difficult (via mid-line laparotomy). Some cases have concurrent sternal deformities.