Proceedings of the 4\textsuperscript{to} Congreso ECVECCS Emergencia y Cuidados Críticos Veterinarios

Nov. 18-20, 2014

Salinas, Ecuador
Pericardial Diseases

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Introduction

The pericardium functions to limit acute cardiac dilatation, maintain cardiac geometry and ventricular compliance, reduce friction, provide a barrier to inflammation from contiguous structures, and buttress the atria. A small amount of fluid is normally found between the epicardial (visceral pericardial) and the parietal pericardial membranes. The term pericardial effusion indicates excessive or abnormal accumulation of pericardial fluid has developed within this space. Pericardial effusion (PE) or constriction (of the heart) may impair cardiac function by interfering with normal cardiac filling. When the heart is compressed, intrapericardial pressure rises, and cardiac filling is impaired, a state of cardiac tamponade is said to be present. Congenital hernias and acquired pericardial effusion are the most clinically relevant disorders of the pericardium in small animal practice. Most cases of PE in dogs are either idiopathic or due to cardiovascular-related neoplasia.

Peritoneopericardial Diaphragmatic Hernia

Important congenital forms of pericardial disease in dogs and in cats include congenital peritoneopericardial diaphragmatic hernia (PPDH) and the rare congenital cysts. Hernias are more common in cats and also develop in dogs (Weimaraner dogs predisposed). Careful radiographic examination leads one to suspect the diagnosis. One typically observes altered radiographic density in the caudoventral portion of the pericardial space. Ventral to the caudal vena cava there may be a persistent mesothelial remnant indicating the dorsal border of the hernia. Often the carina is displaced cranial relative to the caudal border of the heart. Ultrasonography or a barium swallow (if intestinal loops are present) is diagnostic studies. Cardiac tamponade is an infrequent complication and urgent care is more likely to become necessary should there be entrapment of a loop of bowel or strangulation of the liver. The condition is treated surgically, but since the hernia is often an incidental finding in mature animals, the situation may not warrant intervention.

Pericardial Effusion – Etiology & Classification

Acquired pericardial effusion, is very common in dogs; it is very uncommon in cats except in cases of congestive heart failure where effusions often accumulate and may disappear with effective treatment for CHF. The pericardial fluid is typically classified using traditional clinical pathologic methods (the physical description, protein concentration, cell count and distribution, and cytologic features of the fluid). This classification includes transudates, exudates, hemorrhage, and chyle. 

Transudates – in the pericardial space occur secondary to impaired lymphatic or venous drainage of the space or increased permeability of capillaries. Clinical causes include right-sided CHF, peritoneopericardial diaphragmatic hernia, pericardial cysts, hypoalbuminemia, or infections/toxemia (or other causes of increased vascular permeability). These accumulations tend to be necropsy or ultrasound findings and rarely impair heart function.
There are two noteworthy clinical exceptions. Mass lesions at the heart base can obstruct lymphatic drainage leading to a large and compressive, but clear pericardial effusion.

Exudates – in the pericardial space are caused by infective or non-infective pericarditis. This is the classic form of pericardial effusion associated with perforating foreign bodies. While infective pericarditis is not common in small animals, none etiologies include Nocardia infection, migrating plant awns, coccidiomyocosis (Valley Fever), and opportunistic fungi in immunosuppressed dogs (e.g. aspergillosis). Idiopathic, sterile (inflammatory) pericarditis can develop occasionally in the dog and may follow recurrent, idiopathic intrapericardial hemorrhage.

Hemorrhage – into the pericardial space is common (with or without secondary pericardial reaction) is most common in dogs. The most important reason for hemorrhage in younger dogs (<6 years) is idiopathic pericardial hemorrhage in dogs. In some areas, golden retrievers are predisposed. In older dogs, neoplasia of the heart, heart base, or pericardium frequently leads to a hemorrhagic effusion. Hemangiosarcoma of the right atrium (especially in German shepherd dogs, golden retrievers, miniature Schnauzers, and Labrador retrievers) is very common. This neoplasm can be multicentric, with splenic or hepatic involvement and pulmonary metastasis. The aortic body tumor (chemodectoma or paraganglioma) grows along the heart base (and is especially common in aged brachycephalic breed dogs). Ectopic (heart-base) thyroid carcinoma can cause a large heart base mass that can invade the myocardium. Mesothelioma of the pericardium also occurs but is often a controversial diagnosis in light of so-called pericardial fronds, which can develop in dogs with recurrent pericardial effusion. Metastatic carcinoma to the heart is not commonly recognized but has been identified especially in necropsy studies. Lymphosarcoma of the right atrium and ventricles is the most important cardiac neoplasm in the cat, but is considered a rare cause of pericardial effusion in dogs. Uncommon causes of pericardial hemorrhage include: left atrial rupture in dogs with mitral regurgitation; blunt chest trauma; puncture of the heart (knife, bullet or other missile); coagulopathy (rodenticide intoxication), and complicated thoracocentesis.

Chyle is a very rare fluid type in pericardial effusions.

Pathophysiology

Pericardial effusion leads to clinical signs by compressing the heart. Cardiac tamponade refers to "the decompensated phase of cardiac compression resulting from an unchecked rise in the intrapericardial fluid pressure." The normally negative intra-pericardial pressure becomes positive relative to atmosphere. Cardiac tamponade is the mechanism by which low cardiac output and congestive heart failure (CHF) develop with pericardial effusion. Development depends on the rate of fluid accumulation, not simply the volume of pericardial fluid. With few elastic fibers in the pericardium, intrapericardial pressures can rise rapidly once the elastic limits of the membrane are exceeded. This can develop quickly with sudden hemorrhage into the space. In chronic cases, more volume can be accommodated before intrapericardial pressures rise.

Important pathophysiologic features of cardiac tamponade include: Increased (positive) intrapericardial pressure $\rightarrow$ diastolic collapse of the thin walled right atrium and right ventricle and compression of the proximal vena cava $\rightarrow$ reduced right atrial and ventricular filling $\rightarrow$ decreased ventricular preload $\rightarrow$ decreased stroke volume and cardiac output $\rightarrow$ and arterial hypotension if compensatory mechanisms (sympathetic activity,
vasoconstriction, fluid retention) are insufficient.

The clinical consequences of tamponade are clear: Syncope or sudden death may occur if the systemic hypotension is acute and severe. But given sufficient time, compensatory measures are activated to maintain arterial blood pressure. Compensatory measures include heightened sympathetic discharge, systemic vasoconstriction, renal retention of sodium and water, and elevated venous pressures. Extremely high venous pressures may develop behind the heart. Congestive heart failure, with a predominately right-sided component (ascites, pleural effusion) is the consequence of chronic cardiac tamponade.

Additional hemodynamic features of tamponade include equilibration of diastolic pressures in the ventricles, atria and great veins, and respiratory variation in arterial blood pressure (pulsus paradoxicus). The latter is explained by exaggeration in the respiratory-induced variation of right vs. left-sided cardiac filling (i.e. the right ventricle fills better during inspiration and the left ventricle receives less preload; hence, blood pressure falls during inspiration. Additionally, since the heart is now “encased” within a nondistensible sac, as the right heart is better filled, the ventricular septum bulges into the LV, further reducing LV preload.). Experimentally, coronary perfusion is reduced and ischemic injury can develop.

**Clinical Findings In Pericardial Effusion**

**Signalment and History** – Breed predilections have been noted above. The client complaint may be vague. Syncope and collapse are common with acute cardiac tamponade (e.g., sudden hemorrhage) or after diuretic therapy (and volume depletion) for chronic pericardial effusion. In acute cardiac tamponade, the major clinical signs may be low blood pressure and jugular distension but without any fluid accumulation.

Physical findings vary. Fever or thoracic pain may indicate infection or inflammation within the pericardial space. Evidence of systemic disease, such as lymphosarcoma or hemangiosarcoma of the spleen may be noted during a complete physical examination.

Overt signs of right-sided CHF may be evident in chronic pericardial effusion. The physical examination findings of elevated jugular venous pressure, muffled heart sounds, ascites (with or without pleural effusion), and pulsus paradoxicus should prompt investigation of the pericardial space. If venous distension is missed, an erroneous diagnosis of liver disease or abdominal neoplasia may be entertained.

Arterial hypotension or pulsus paradoxicus may be detected by using a Doppler flow detector and gradually reducing the pressure within the cuff (breakthrough systolic flow signals occur during expiration but are lost during inspiration). The central venous pressure is generally quite high, often exceeding 12 cm H₂O [normal < 5 cm].

Cardiac auscultation is characterized by distant heart sounds. A pericardial friction rub may indicate pericarditis, but this is a rare finding in dogs. Breath sounds are muffled and there will be tachypnea or respiratory distress if there is pleural effusion from CHF.

**Diagnostic Studies** – Routine diagnostic studies are helpful in establishing the diagnosis and recognizing the cause and complications of disease.

An electrocardiogram may show any of the following: Decreased amplitude QRS complexes is the most common (but variable) EKG finding; Electrical alternans occurs with large effusates and swinging of the heart but is not evident in small to moderate effusions; or ST segment elevation is least common, but represents an epicardial injury current with
likelihood of pericarditis. Sinus tachycardia is typical, but vagal reflexes can be invoked that promote sinus arrhythmia or bradyarrhythmias. Atrial and ventricular arrhythmias may be observed secondary to myocardial involvement, ischemia, or concurrent primary heart disease.

**Radiography** generally demonstrates abnormalities once there is a significant accumulation of pericardial fluid. The cardiac silhouette enlarges, loses its angles and waists, and eventually becomes globular in shape ("basketball or soccer ball heart"). The left atrial border on the lateral view may become very rounded. The cardiac outline often becomes sharp, presumably from diminished motion at the periphery of the silhouette. Should a metallic foreign body be observed over the heart on two views, constrictive or constrictive-effusive disease is likely. Metallic densities in the pericardium (such as a shotgun pellet) should be taken as risk factors for pericarditis, even if not currently evident in the cardiac region. Pulmonary vascularity is often reduced from low cardiac output (in contrast to CHF from cardiomyopathy or valvular disease).

If CHF has developed, there may be increased pulmonary interstitial densities (edema), distension of the caudal vena cava, hepatomegaly, or pleural effusion. Heart base tumors may deviate the trachea (generally to the right and dorsad), producing a mass effect. Fluoroscopy may demonstrate reduced cardiac motion. Pneumopericardiography can identify intrapericardial mass lesions, but is rarely done, as echocardiography is safer.

**Echocardiography** is a highly sensitive test for detecting pericardial effusion. Abnormal fluid accumulation is evident as a sonolucent (generally black) space between the epicardium and pericardium, extending from apex to base. A mixed intrapericardial echogenic pattern suggests cellular exudate or recent hemorrhage. The effusion may be loculated (localized) in inflammatory diseases. Cardiac mass lesions may be observed, especially in dogs. A tumor of the right atrium or along the right atrioventricular groove suggests hemangiosarcoma. A heart base mass around the aorta is typical of chemodectoma or ectopic thyroid carcinoma. Thickened pericardium with tumors on the parietal surface may suggest mesothelioma. In dogs with CHF, pleural effusions and dilated caudal vena cava and hepatic veins also may be observed. The recognition of diastolic collapse of the right atrium or right ventricular wall is supportive of increased intrapericardial pressure and corresponds to effusion with tamponade. Inversion of the atrial wall or protracted collapse of the wall is a more specific sign. There are however both false positives (occasionally pleural effusion causes this in dogs) and false negatives (if there is concurrent right sided CHF with elevated CVP expanding the cardiac chambers).

In older dogs, the distinction between idiopathic hemorrhagic pericardial effusion and bleeding from a tumor is crucial in terms of prognosis. This may not be possible without a high resolution, technically proficient, echocardiogram recorded from each side of the thorax and using multiple angled views.

In some cases, exploratory surgery or advanced imaging (CT, MRI) is needed to exclude a mass lesion. In one small canine study, cardiac MRI did not substantially improve the diagnostic yield for cardiac masses.

**Clinical laboratory** evaluation is may simply reflect the consequences of heart failure or prior diuretic therapy. The CBC may indicate inflammation, infection, or hemorrhage. Increased numbers of circulating nucleated RBC’s are suggestive of hemangiosarcoma of the spleen (and heart) in dogs with pericardial effusion. Analysis of pleural or peritoneal
effusions generally indicates fluid of obstructive origin (transudate, modified transudate, or infrequently chyle). Bacterial cultures of the effusate or serum fungal titers may be positive when pericarditis is related to these infections. Cardiac troponins may increase owing to myocardial ischemia or inflammation.

**Cytology & Analysis of Pericardial Fluid** – Fluid can be collected from the pericardial space by pericardiocentesis for laboratory analysis. Prior to fluid removal, measurement of "static" intrapericardial fluid pressure can be accomplished by attaching one end of a saline-filled extension tube to the intrapericardial catheter and the other end to a central venous pressure manometer. This is an optional but instructive procedure. Cases of tamponade demonstrate a high (positive) pressure, usually > 12 cm H₂O above the midsternal line, with the patient resting in lateral recumbency. The pressure becomes subatmospheric following pericardiocentesis and rises with ventilation. With constrictive-effusive pericardial disease (pericardial effusion with a thick pericardium but without tamponade), the intrapericardial pressure is closer to normal (i.e., near zero cm H₂O). While this relatively crude method does not precisely measure intrapericardial pressure, it can offer useful clinical information. Although rarely done, a sample also can be saved anerobically for determination of pH of pericardial fluid. If the value obtained is < 7.0, this finding is suggestive of pericardial inflammation or idiopathic hemorrhage, though more data are needed to precisely define the predictive value of this finding. Values of 7.35 or greater are more suggestive of neoplasia or recent hemorrhage.

Collected fluid can be analyzed and classified as a transudate, exudate, hemorrhage, or chyle (see above). Unfortunately, except in cases of lymphosarcoma or septic inflammation, cytologic examination may not be especially helpful. It can be difficult to conclusively identify neoplastic cells within pericardial effusates. The problems include poor exfoliation or overinterpretation of reactive mesothelial cells.

**Therapy & Prognosis**

The treatment of choice for initial stabilization of the dog with cardiac tamponade is pericardiocentesis. Not all of the fluid must be removed to obtain benefit since the intrapericardial pressure usually falls dramatically once ½ of the volume is removed. The general procedure used by the author is described below for your reference.

**Pericardiocentesis** can be accomplished using a needle, butterfly infusion needle (for very small dogs), a through-the-needle catheter, an over-the-needle catheter, a commercial thoracocentesis trocar system, a chest drain (for large animals), or a balloon dilation catheter (which can be used to rip the pericardium). This procedure is most often performed in dogs, for which we prefer a 14 to 16 gauge Angiocath®, over-the-needle catheter. Generally, an IV should be placed for emergencies or for volume loading should hypotension develop. If arterial blood pressure is stable, mild sedation is often tolerated and improves the procedure for all (for dogs, butorphanol 0.1 to 0.25 mg/kg, IM). Should hypotension develop after sedation, quickly infuse saline solution intravenously and give atropine if the heart rate has decreased.

The tap can occur from either the right side (cardiac notch) or left hemithorax (depending on preference, the situation, radiography, and echocardiography). We prefer the right-sided intercostal approach to avoid the largest coronary vessels. The small animal patient is
placed in lateral recumbency and the spine elevated slightly with a radiographic foam wedge. ECG leads are attached. The needed depth of penetration and the ideal puncture site can be guided by echocardiography (to simply identify the largest effusion space); alternatively, one can note the strongest palpable cardiac impulse (both points are usually the same). Placing the patient in a slightly oblique position, and later rotating the animal as needed, facilitates fluid withdrawal and patient restraint during the procedure. Following scrubbing of the skin, a local 2% lidocaine block of the skin, subcutaneous tissues, pleura, and superficial pericardium (if possible) is made with a 25- or 23- gauge needle. In small dogs the small gauge needle may transiently enter the pericardial space, providing some guide to the depth needed for catheter placement.

After gloving for the procedure, the operator prepares sterile equipment. When using an over the needle catheter (Angiocath®), an additional side-hole is cut into the edge of the catheter and the needle stylet is replaced. Alternatively, a commercial canine pericardiocentesis catheter can be used if available. The catheter is then advanced through the skin, subcutaneous tissue, and intercostal space in a controlled but smooth motion and inserted deliberately into the pericardial space. The ECG is monitored for extrasystoles in case the heart is pricked with the catheter (note: dorsal approaches may cause the catheter to perforate the atrium and this will not be associated with premature ventricular beats). Once the pericardial space is entered, fluid (usually bloody) enters the catheter lumen. If intrapericardial pressure will be measured, the line connecting the CVP-manometer is immediately attached to the catheter because the intrapericardial pressure rapidly declines with aspiration of the fluid. After measuring intrapericardial pressure (optional), fluid samples are collected for cytology (EDTA and plain tubes) and culture (pending cytology).

The effusion is now drained as completely as possible. Owing to the relatively inelastic properties of the pericardium, the removal of even modest amounts of effusate may be very beneficial. The effects on the patient are often dramatic with a marked improvement in attitude, color, and peripheral pulse pressure. When complete, the aspirated fluid volume is quantified. Repeated intrapericardial pressure measurement and/or echocardiography can be used to verify the benefit of the procedure if desired. A sample of the effusate is evaluated by microscopy for cellular abnormalities and bacteria, and is then cultured (aerobic, anaerobic) if appropriate.

In patients with culture negative, idiopathic pericardial hemorrhage (or idiopathic pericarditis), conservative treatment with catheter drainage, may be “curative,” though diligent follow-up (for at least one year) is needed to assure that constrictive pericardial disease does not develop. In cases related to neoplasia, or in cases of recurrent pericardial effusion, this treatment must be repeated or another surgical approach taken (see below).

**Medical therapy** is not a prominent feature of this disorder. Intravenous saline, at shock doses, may be needed in cases of hypotension due to severe or sudden cardiac tamponade. While furosemide and venodilators can decrease elevated venous pressures, *they are not substitutes for pericardiocentesis* in the symptomatic patient; furthermore, these drugs may reduce ventricular filling predisposing to hypotension. In general, diuretics are contraindicated except in recurrent, neoplastic-related right-sided CHF in which venous pressures can become exceptionally high.

Following successful pericardiocentesis, and in patients with large fluid accumulations, it is appropriate to administer a single dose of 1 mg/kg furosemide SQ to enhance renal excretion of sodium (and overcome the sodium-retaining consequences of cardiac tamponade that often persist for some time following pericardiocentesis or pericardiectomy).
Even without diuretics, urine flow increases rapidly, often resulting in voiding on the pericardiocentesis table!

Other Treatments – Empirical use of antibiotics and of corticosteroids has offered no certain benefit in cases of idiopathic pericardial hemorrhage and are no longer used by us. Drugs that prevent fibrosis might be considered, but these have not been suitably investigated. Antineoplastic drugs have provided generally poor results in patients with cardiac tumors (aside from lymphoma), though there are exceptional cases. Specific causes of pericardial infection - e.g. coccidiomyocosis or lymphoma - have specific adjunctive treatments. Radiation therapy can be used to reduce the size of heart base mass lesions, but there are issues related to prognosis and collateral tissue injury. Only limited case reports have been presented thus far.

Thoracocentesis is a helpful adjunct in large pleural effusions. Ascitic effusions need not be tapped if pericardiocentesis is performed.

Surgical Therapy may be necessary for successful management of pericardial diseases in the following situations.

Diagnosis – Surgical biopsy of the pericardium may be needed to exclude a diagnosis of mesothelioma, as this neoplasm is not readily visualized by echocardiography unless large tumors are generated. Surgery or thoracoscopy may also provide additional visualization of the heart base or auricular area and identify a neoplasm that might have been missed during echocardiography. In the older dog with PE from undefined cause, this is both a diagnostic and therapeutic procedure in many cases.

Idiopathic hemorrhage – Subtotal pericardiectomy (ventral to the phrenic nerves) may be needed in recurrent idiopathic hemorrhagic effusion. A surgical approach seems preferable as more of the pericardium can be removed and these dogs are often young and relatively healthy. Either a sternotomy or thoracotomy approach can be taken. The efficacy of pericardial windows for this condition has not been sufficiently evaluated by long term follow up (i.e. for > 2 years).

Pericarditis – The treatment for infective, suppurative pericarditis in small animals is specific antibiotic therapy based on aerobic and bacterial anaerobic culture, initial catheter drainage of the pericardium, and subsequent surgical removal of as much of the pericardium as possible (to prevent pericardial constriction – see below). This requires a thoracotomy or sternotomy and subsequent placement of one or more thoracic drain tubes to treat the resultant pyothorax. A foreign body should be sought in these cases.

Constrictive Pericardial Disease – Surgery is indicated if constrictive (effusive) pericarditis is diagnosed, as with chronic coccidiomyosis infection in dogs. The only effective therapy is surgical removal of the pericardium ventral to the phrenic nerves. Mortality rate is high.

Cardiac neoplasia in dogs – In dogs with defined neoplasia, a palliative pericardial “window” can be created to prevent recurrent tamponade (though there is also a risk of uncontrolled bleeding, especially with hemangiosarcoma). This procedure can be performed by mini-thoracotomy with long instruments (such as those used for laparoscopy) or via a subxiphoid approach. Thoracoscopy also has been used to create pericardial windows and may be is a reasonable palliation for neoplasia-associated pericardial effusion as well as initial treatment for debilitated dogs with infective pericarditis (later perform full pericardiectomy).
**Prognosis** – The prognosis of pericardial disease depends on the cause, but is generally favorable with idiopathic hemorrhagic pericardial hemorrhage in dogs, guarded with infective pericarditis, and unfavorable with cardiac or heart base neoplasia (unless there is good surgical access for removal, which is rarely the case). While a chemodectoma grows slowly (often with a one year survival), right atrial hemangiosarcoma is often metastasized by the time of diagnosis. Ectopic thyroid carcinomas can be particularly invasive. Mesothelioma can spread to the pleural space.

**Constrictive Pericardial Disease**

**Definition** – Constrictive pericarditis can be likened to a "shrink wrap" or concrete envelope that envelops the heart and limits overall cardiac volume. The condition typically follows chronic inflammation. As the intrapericardial fluid is reabsorbed, the pericardial space becomes obliterated. The pericardium thickens and scars, the ventricles become constricted, and ventricular filling is limited. The result is reduced preload. Constrictive pericardial disease eventually leads to limited cardiac output and to right-sided (or biventricular) CHF. The term "constrictive-effusive" pericarditis refers to a state of constrictive physiology with concurrent pericardial effusion (but without tamponade). This is particularly important in the dog.

**Causes** – Pericardial constriction most often develops secondary to chronic inflammation consequent to infective or idiopathic pericarditis or recurrent hemorrhage. Rarely, the pericardium may undergo calcification from chronic inflammation.

**Clinical Features** – Most dogs are presented with signs of right-sided CHF (ascites or pleural effusion) with jugular venous distension. A gallop sound termed a pericardial “knock” may be heard and is an indicator of abrupt restriction to ventricular filling. Disparate rates of cardiac filling are also reflected by prominent and multiple diastolic movements of the ventricular septum on 2D echocardiography and dramatic variations in transvalvular inflow related to the phase of ventilation. A tall and brief E-wave may be seen on the Doppler Echo, indicating sudden termination of filling. Imaging of cardiac size may not reveal dramatic enlargement owing to constriction by the thickened pericardium. Echocardiography is helpful in the diagnosis and shows right atrial dilation and Doppler changes described above. But diagnosis by Echo usually requires referral to a cardiac specialist as findings can be confusing. Cardiac catheterization of the right heart can be used to identify characteristic pressure tracings. MRI is very useful in people.

The treatment of choice for constriction is surgical removal of the pericardium and possible decorticating of the epicardium. This carries a significant surgical and perioperative risk.

Additional Reading