Pericardial effusion in dogs: Differentials and management

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Pathophysiology:

Pericardial effusion (PE) is the accumulation of excessive fluid in the pericardial sac. Because the flow of blood is dependent on a pressure gradient, increasing the external pressure on the heart will cause collapse of the lower-pressure chambers (RA, LA, and in extreme cases, RV), resulting in tamponade. This prevents blood from flowing back into the heart from the venous circulation, which results in both obstructive shock from lack of preload entering the heart, as well as signs consistent with right sided heart failure (venous engorgement, pleural and peritoneal effusions, hepatic engorgement) due to increased venous filling pressures as the blood backs up. Acute PE tends to be smaller volume, higher pressure, and present with more acute signs of shock. Chronic PE tends to be larger volume and lower pressure as the pericardium has had time to stretch. It is also often associated with more severe pleural/peritoneal fluid volumes due to increased fluid retention over time as the body tried to compensate for low cardiac output. Not all cases of PE result in tamponade; depending on the amount of fluid and pressure exerted on the heart chambers, clinical signs of PE can range from asymptomatic to severe shock and death.

Diagnosis:

PE is often initially suspected based on history/signalment and physical exam. As the most common causes of acute effusions are neoplastic, animals are often older and present with a history of acute collapse or sudden worsening lethargy and pallor. More chronic effusions may present for abdominal distention or respiratory distress secondary to cavitary fluid accumulation. In a recent study by Fahey (JVECCS 2017), about 50% of dogs with PE presented with a history of vomiting within the previous 48 hours. Physical exam findings may include muffled heart sounds, jugular distention, dyspnea, abdominal distention secondary to ascites, and perfusion parameters consistent with hypodynamic shock.

Pulsus paradoxicus is commonly described in association with tamponade, but can be difficult to appreciate clinically, especially if pulse quality is poor or the patient is panting. It is an exaggeration of the normal change in pulse pressure associated with respiration (in health, too slight to feel) whereby negative pressure on inspiration augments right sided volume and pulmonary vascular filling at the expense of left sided cardiac filling, and reverses during expiration. This results in pulses that are stronger on expiration and weaker on inspiration.

Electrical alternans, also commonly associated with pericardial effusions, is a different phenomenon as it is an ECG finding that does not affect cardiac output or pulse pressure. It is more commonly seen in larger volume effusions where swinging of the heart in the pericardial sac results in cyclical variation in R wave height, usually in a 1:1 or 2:2 ratio. Other common EKG findings include sinus tachycardia and ventricular arrhythmias, particularly during pericardiocentesis.

Thoracic imaging is necessary for diagnosis of PE. While radiographic findings such as an enlarged globoid heart +/- evidence of thoracic and/or abdominal effusion are consistent with PE, they are fairly insensitive (sensitivity 41.9%, specificity 40% Cote JAVMA 2013) in differentiating it from other causes of heart disease. However, radiographs are often still performed to look for metastatic disease. Trans-thoracic echocardiography remains the gold standard for diagnosis of PE, and can be performed cage-side with minimal training. Detection of the underlying cause of the effusion (e.g. is there a mass?) is more difficult and is often best performed by an experienced echosonographer, ideally prior to pericardiocentesis if patient stability permits (the presence of fluid aids in visualization of some masses, depending on location). Compared to surgical or necropsy findings, ultrasound has variable sensitivity for cardiac masses (60-95%), but is very specific (98-100%) (Yamamoto J vet Med Sci 2013, MacDonald JAVMA 2009). Advanced imaging (CT, MRI) are equivalent to echo in detection of cardiac masses (Scollan JVIM 2015, Boddy JVIM 2011); while they allow for screening for masses and metastasis using only one modality, they also must be balanced against the need for sedation/anesthesia to perform them in a possibly unstable patient, as well as cost and availability limitations.

Once PE is identified with ultrasound, pericardiocentesis is both therapeutic and potentially diagnostic. Pericardiocentesis should be performed in the 5-6th intercostal space (where the elbow hits the chest) on the ventral right side of the thorax to avoid the lungs and coronary vessels. PE is almost always hemorrhagic and, while it is never wrong to submit the fluid for cytology, hemorrhagic PE is usually non-diagnostic on cytology. Non-hemorrhagic fluids should always be submitted for analysis; for hemorrhagic appearing samples, in a JVIM 2013 paper by Cagel et al, diagnostic yield increased 3-fold if the PCV of the sample was <10%. Therefore, even if PE appears grossly hemorrhagic, at minimum the PCV of the sample can be quickly and inexpensively assessed; if lower than expected, the fluid should be submitted for full analysis. Infectious/inflammatory causes and tumors such as lymphoma that may exfoliate into effusions can often be diagnosed from fluid analysis, but the more common causes of PE (hemangiosarcoma, chemodectoma, mesothelioma, and idiopathic effusions) usually cannot.

Various biomarkers and analyses of PE have been explored to see if there are factors in the fluid that could help differentiate neoplastic from non-neoplastic causes. While sometimes statistically different, effusion and plasma pH, electrolyte concentrations, glucose, lactate, troponins, and NT-proBNP have all failed to demonstrate enough clinically significance difference between neoplastic and non-neoplastic causes of PE. In cases of “idiopathic” PE, PCR evaluation in 16 of 68 dogs around the Mediterranean region were positive for vector-borne pathogens including leishmaniasis, anaplasma, babesia, and hepatozoonosis; whether these organisms were causal of the effusion is unknown (Tabar JSAP 2018). In another study, viral and bacterial DNA in 10 dogs with PE found influenza A in one sample, but no other positive results (Zini Vet J 2009). Infectious etiologies are causes of PE in humans, so further research is needed as to whether some causes of “idiopathic” canine PE may truly have an underlying infectious etiology.

Location and appearance of mass lesions on echo are often used to weight likely differentials, but is only moderately accurate (50-78%); definitive diagnosis of cardiac mass lesions requires some form of FNA or biopsy if fluid cytology is non-diagnostic. While not commonly performed, FNA of cardiac masses (if location permits) has been reportedly successful at obtaining a diagnosis in a small number of dogs (n=6) (Perdo J vet cardio 2016) with no complications. More commonly, if a definitive diagnosis is desired, biopsies are attained at time of palliative pericardectomy or necropsy.

Differentials:

Common causes of PE in dogs include neoplastic and idiopathic effusions, with infectious/inflammatory, toxic, traumatic, or congenital causes less commonly observed. While obviously not absolute, in general, younger animals tend to have congenital (e.g. PPDH), toxic, or infectious causes, middle aged dogs tend to have idiopathic, infectious/inflammatory, toxic (e.g. anticoagulant rodenticides), or foreign body causes, and older dogs tend to have neoplastic causes of effusions. Dogs with a history of heart disease can also have left atrial rupture as a cause for hemorrhagic PE. CHF can also result in a more transudative PE in dogs, although this is more typical for cats. Other systemic diseases may also cause PE, such as severe uremia, SIRS, severe hypoalbuminemia, etc., but these do not usually cause tamponade and often resolve when the underlying systemic disease is addressed.

The most common type of cardiac neoplasia in dogs is hemangiosarcoma, which accounts for about 69% of all cardiac tumors in dogs. These tumors are most commonly located at the right AV groove or right auricle. Other common tumors include aortic body tumors (chemodectoma, paraganglioma) and ectopic thyroid carcinoma, which are more commonly located at the heart base. Lymphoma and mesothelioma can vary widely in appearance and location or have no visible mass lesions on echo if the disease is diffuse. Other types of primary and secondary cardiac neoplasia have been sporadically reported in the literature but are rare.

In a retrospective study by Boston et al JAAHA 2011, of 23 dogs presenting with splenic hemangiosarcoma as the reason for work-up, 9% had a concurrent cardiac mass. Of 31 dogs presenting with PE and cardiac hemangiosarcoma as the presenting complaint, 42% had evidence of metastasis at another anatomic site. In a study by Yamamoto (J vet med sci 2013), of 51 dogs with cardiac hemangiosarcoma, 75% had metastasis at time of necropsy. In another study (MacDonald JAVMA 20009), all neoplastic causes of pericardial effusion had metastatic rates of 50-66%. Therefore, if owners want to proceed with therapy beyond palliative pericardiocentesis for PE with an underlying cardiac mass, full staging is recommended.

Prognosis and therapeutic options:

In the short term, pericardiocentesis is the definitive life-saving therapy needed for any patient in tamponade showing signs of shock. Fluid therapy can also help augment cardiac filling in the stabilization process, but will contribute to third spacing of fluid if pericardiocentesis is not also performed within a short time frame.

Long-term prognosis for PE is extremely variable depending on cause. Diseases that are treatable (infectious, congenital, FB, toxin, etc.) can have an excellent prognosis if cure can be achieved, but as some infectious causes (fungal, protozoal, etc.) may be difficult to resolve, the prognosis can range from guarded to excellent depending on etiology. Many cases of infectious pericarditis will benefit from pericardectomy, not only to allow surgical debridement and definitive diagnosis of the causative process, but to prevent constrictive pericarditis which can develop over time after any chronic cause of inflammatory pericardial effusion.

Idiopathic PE is a rule-out diagnosis, and is often suspected to have an underlying immune-mediated or infectious cause that is simply not found. It can also have an excellent prognosis if the effusion is self-

limiting or curative pericardectomy is performed. Survival rates of 72% at 18 months and 50% at 1500 days after diagnosis have been reported. Therefore, clinicians should be cautious of "doom and gloom" prognoses for hemorrhagic pericardial effusions if an underlying neoplastic process is not seen on serial imaging, especially for younger to middle-aged animals.

Overall prognosis for neoplastic effusions where owners do not wish to perform further therapy beyond palliative pericardiocentesis is generally poor, with median survival times of 7.1 days (1-26d). However, depending on underlying type of neoplasia, for owners who want to pursue additional therapy, the survival times can vary widely depending on tumor type.

For tumors whose location is amenable to surgical resection (generally the right auricular appendage), thoracotomy or thorascopic surgery + pericardectomy can be diagnostic as well as therapeutic. While pericardectomy alone will not stop pericardial effusion from forming with non-resectable tumors, the ability of the fluid to exit into the pleural space will prevent the life-threatening tamponade that is the most serious sequella of the effusion.

Heart-based tumors of neuroendocrine origin are often slow-growing and late to metastasize, so palliative therapy with pericardectomy can significantly increase survival times in these patients. In a 2001 JAVMA paper by Vicari, dogs without pericardectomy had a median survival time of 129 ± 51 days, while dogs who underwent pericardectomy had a significantly longer median survival time of 661± 170 days. Similarly, mesothelioma treated with pericardectomy has a reported median survival time of 13.6 months, with 80% of patients surviving 1 year and 40% surviving to 2 years. Radiation therapy for a heart based tumor has also been described by Rancillio (JAVMA 2012).

Hemangiosarcoma, which is the most common cardiac tumor, also unfortunately has the worst prognosis, with average survival being 1-3 months. Most dogs whose owners do not seek invasive therapy are euthanized within 30 days of diagnosis. Yunnan Baiyao and aminocaproic acid have been used palliatively to try to reduce reformation of hemorrhagic PE; while they have not caused harm, they also were not shown to delay recurrence of clinical signs in a cohort of 67 dogs (Murphy JVECCS 2017). In a small retrospective of 23 dogs who all underwent surgical resection of cardiac hemangiosarcoma, dogs that received chemotherapy had a statistically longer MST (mean 164 days, range 36-229) compared to those that received surgery alone (mean 46, range 0-138d). In a study by Ghaffari (JSAP 2014), dogs receiving doxorubricin based chemotherapy protocols for RA masses with effusion had a MST of 140d (2-302 days) without surgery. In 6 dogs with presumptive hemangiosarcoma as a cause of PE, a single dose of radiation was safe and reduced frequency of palliative pericardiocentesis with a MST of 79 days (Nolan J vet cardio 2017). Whether these gains in survival times are worth the invasiveness and expense of each intervention is a personal decision that each client must make for each individual pet after consultation with their veterinarian.