

CHAPTER 45

PERICARDIAL DISEASES

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KEY POINTS

- Pericardial effusion is the most common pericardial disorder.
- Most pericardial effusions in dogs are hemorrhagic and of neoplastic or idiopathic origin.
- Hemangiosarcoma is by far the most common neoplasm causing pericardial effusion in dogs.
- Cardiac tamponade occurs when intrapericardial pressure rises to equal or greater than normal cardiac filling pressure.
- The rate of pericardial fluid accumulation influences how quickly cardiac tamponade develops; a large pericardial fluid volume implies a gradual process.
- Clinical signs usually reflect poor cardiac output and systemic venous congestion.
- Echocardiography is the main clinical tool for diagnosing pericardial effusion and cardiac masses.
- Right atrial collapse is a characteristic echocardiographic finding with cardiac tamponade.
- Immediate pericardiocentesis is indicated for cardiac tamponade.

The pericardium is a closed serosal sac that envelops the heart and is attached to the great vessels at the heart base. It provides a barrier to infection and inflammation from adjacent tissues and helps balance the output of the right and left ventricles. A small amount (approximately 0.25 ml/kg body weight) of clear, serous fluid normally serves as a lubricant between the visceral pericardium (epicardium), which is directly adhered to the heart, and the outer fibrous, parietal pericardial layer.¹

The most common pericardial disorder involves excess or abnormal fluid accumulation within the pericardial sac (pericardial

effusion). This occurs most often in dogs and can lead to signs of severe cardiac dysfunction. Other acquired and congenital pericardial diseases are seen infrequently. Acquired pericardial disease that causes clinical signs is uncommon in cats.²

PERICARDIAL EFFUSION

Hemorrhagic Pericardial Effusion

Most pericardial effusions in dogs are serosanguineous or sanguineous. The fluid usually appears dark red, with a packed cell volume (PCV) more than 7%, a specific gravity greater than 1.015, and a protein concentration greater than 3 g/dl. The underlying etiology usually is either neoplastic or idiopathic. Neoplastic effusions are more likely in dogs older than 7 years of age.³

Other, less common causes of intrapericardial hemorrhage include left atrial rupture secondary to severe mitral insufficiency, coagulopathy (especially from rodenticide toxicity or disseminated intravascular coagulation), and penetrating trauma.

Hemangiosarcoma

Hemangiosarcoma (HSA) is by far the most common neoplasm causing hemorrhagic pericardial effusion in dogs; it is rare in cats. Most HSAs arise in the right atrium or right auricle, but some also infiltrate the ventricular wall.^{4,5} Occasionally, HSA occurs in the left ventricle, in the septum, or at the heart base. Metastases are common by the time of diagnosis.

Heart base tumors

The most common heart base tumor is the chemodectoma (aortic body tumor), which arises from chemoreceptor cells at the base of

the aorta.⁴ Thyroid, parathyroid, lymphoid, and connective tissue neoplasms also can occur at the heart base. Heart base tumors tend to be locally invasive around the root of the aorta and surrounding structures; however, metastases to other organs can occur.⁶

Other neoplasia

Hemorrhagic pericardial effusion also can occur with pericardial mesothelioma, malignant histiocytosis, some cases of cardiac lymphoma, and occasionally with metastatic carcinoma. Pericardial mesothelioma may cause mass lesions at the heart base or elsewhere but often has a diffuse distribution and may mimic idiopathic disease.^{7,8} Lymphoma involving various parts of the heart is more common in cats than in dogs (and often causes a modified transudative effusion). Other primary tumors of the heart are rare but include myxoma, and various types of sarcoma.

Idiopathic (benign) pericardial effusion

Idiopathic pericardial effusion is also a relatively common cause of canine hemorrhagic pericardial effusion. It is reported most often in medium to large breed, middle-aged dogs. More cases have been reported in males than females. Its cause is still unknown; however, mild pericardial inflammation, with diffuse or perivascular fibrosis and focal hemorrhage, are common histopathologic findings.⁹⁻¹¹

Transudative Pericardial Effusion

Transudates or modified transudates occasionally accumulate in the pericardial space of both dogs and cats. A chylous effusion occurs rarely. Pure transudates are clear, with a low cell count (usually <1000 cells/ μ l), specific gravity (<1.012), and protein content (<2.5 g/dl). Modified transudates may appear slightly cloudy or pink tinged. Their cellularity (approximately 1000 to 8000 cells/ μ l) is still low, but total protein concentration (approximately 2.5 to 5 g/dl) and specific gravity (1.015 to 1.030) are higher than those of a pure transudate.

Transudative effusions can develop with congestive heart failure (CHF), hypoalbuminemia, congenital pericardial malformations, and toxemias that increase vascular permeability (including uremia). These conditions usually are associated with relatively small-volume pericardial effusion, and cardiac tamponade is rare. In cats, pericardial effusion is most commonly associated with CHF from cardiomyopathy.¹² Effusion associated with cardiac lymphoma also often appears transudative.

Exudative Pericardial Effusion

Exudative effusions are cloudy to opaque or serofibrinous to serosanguineous. They typically have a high nucleated cell count (usually much higher than 3000 cells/ μ l), protein content (often much above 3 g/dl), and specific gravity (>1.015). Cytologic findings are related to the etiology. Exudative pericardial effusions are uncommon in small animals. However, they have occurred from plant awn migration, extension of a pleural or mediastinal infection, and bite wounds. Various bacteria (aerobic and anaerobic), actinomycosis, coccidioidomycosis, aspergillosis, disseminated tuberculosis, and, rarely, systemic protozoal infections have been identified. Feline infectious peritonitis is the most important cause of symptomatic pericardial effusion in cats. Exudative effusions also have occurred in association with leptospirosis, canine distemper, and idiopathic pericardial effusion in dogs. Chronic uremia occasionally causes a sterile, serofibrinous or hemorrhagic effusion.

CARDIAC TAMPONADE

The development of clinical signs from pericardial effusion is associated mainly with increased intrapericardial pressure. Because the

fibrous pericardium is relatively noncompliant, increases in fluid volume can sharply raise intrapericardial pressure. *Cardiac tamponade* develops when intrapericardial pressure rises toward and exceeds normal cardiac diastolic pressures.^{3,13} The external cardiac compression progressively limits right ventricular filling and, with increasing severity, also reduces left ventricular filling.^{3,13} Systemic venous pressure increases and forward cardiac output falls. Eventually, diastolic pressures in all cardiac chambers and great veins equilibrate.^{3,13}

The rate of pericardial fluid accumulation and the distensibility of the pericardial sac determine whether and how quickly cardiac tamponade develops. Rapid accumulation of a relatively small-volume effusion (e.g., 50 to 100 ml) can raise intrapericardial pressure markedly because pericardial tissue stretches slowly. Conversely, a slow rate of fluid accumulation may allow for enough pericardial distension to maintain low intrapericardial pressure until the effusion is quite large. A large volume of pericardial fluid implies a gradual process. So long as intrapericardial pressure is low, cardiac filling and output remain relatively normal and clinical signs are absent. Fibrosis and thickening further limit the compliance of pericardial tissue, and this can increase the likelihood of cardiac tamponade.

Neurohormonal compensatory mechanisms are activated as cardiac output falls.^{14,15} These contribute to fluid retention and other clinical manifestations of tamponade. Signs of systemic venous congestion become especially prominent over time. Although pericardial effusion does not directly affect myocardial contractility, reduced coronary perfusion during tamponade can impair both systolic and diastolic function. Low cardiac output, arterial hypotension, and poor perfusion of other organs besides the heart can ultimately precipitate cardiogenic shock and death.

Cardiac tamponade also causes an exaggerated respiratory variation in arterial blood pressure known as *pulsus paradoxus*. Inspiration normally lowers intrapericardial and right atrial pressures slightly, which enhances right-sided heart filling and pulmonary blood flow. Left-sided heart filling diminishes as more blood is held in the lungs and the inspiratory increase in right ventricular filling pushes the interventricular septum leftward. Thus left-ventricular output and systemic arterial pressure normally decrease slightly during inspiration. Pulsus paradoxus is an exaggeration of this normal pressure difference with respirations; patients with pulsus paradoxus exhibit a fall in arterial pressure during inspiration of 10 mm Hg or more.^{16,17}

CLINICAL PRESENTATION

Cardiac tamponade is relatively common in dogs but rare in cats. Clinical findings reflect poor cardiac output and usually systemic venous congestion as well. The typical history includes exercise intolerance, abdominal enlargement, tachypnea, weakness, collapse or syncope, and sometimes cough. Collapse is more common in dogs with cardiac neoplasia than in those with idiopathic disease.¹⁸ Nonspecific signs such as lethargy, inappetence, or other gastrointestinal maladies can develop before obvious ascites does. Cases with pericardial effusion but without cardiac tamponade may show signs of the underlying disease process or be asymptomatic.

Physical Findings with Tamponade

Jugular venous distention or a positive hepatojugular reflux,* hepatomegaly, ascites, labored respiration, and weakened femoral pulses

*The hepatojugular reflux is assessed by applying firm pressure to the cranial abdomen while the animal stands quietly with head in a normal position. This pressure transiently increases venous return, but normally there is little to no change in jugular vein appearance. Jugular distention that persists while abdominal pressure is applied constitutes a positive (abnormal) test result.

are common physical findings.^{3,13,18-21} Pulsus paradoxus is detected occasionally by femoral pulse palpation. High sympathetic tone commonly produces sinus tachycardia, pale mucous membranes, and prolonged capillary refill time. The precordial impulse is palpably weak with a large pericardial fluid volume, and heart sounds are muffled by moderate to large pericardial effusions.^{3,18,19} In addition, lung sounds can be muffled ventrally with pleural effusion. Pericardial effusion alone does not cause a murmur, but concurrent cardiac disease may do so. Reduced lean body mass (cachexia) is apparent in some chronic cases.

Although right-sided congestion predominates, signs of biventricular failure can occur. Rapid pericardial fluid accumulation can cause acute tamponade, shock, and death without signs of pleural effusion, ascites, or radiographic cardiomegaly. Pulmonary edema, jugular venous distention, and hypotension may be evident in such cases.

DIAGNOSIS

Cardiac tamponade is often suspected from the history and physical examination, but thoracic radiographs and especially echocardiography are important for diagnosis. The electrocardiogram (ECG) may suggest pericardial disease in some cases.

Thoracic Radiographs

The appearance of the cardiac silhouette depends on the volume of pericardial fluid as well as any underlying cardiomegaly.^{3,13,22,23} Massive pericardial effusion causes the classic globoid-shaped cardiac shadow (“basketball heart”) seen on both views. But other causes of a large, rounded heart shadow include dilated cardiomyopathy or marked tricuspid (with or without mitral) insufficiency. Smaller volumes of pericardial fluid allow some cardiac contours to be identified, especially those of the atria.

Other radiographic findings associated with tamponade include pleural effusion, caudal vena cava distention, hepatomegaly, and ascites. Pulmonary infiltrates of edema or distended pulmonary veins are noted only occasionally. Tracheal deviation, a soft tissue mass effect, or metastatic lung lesions are seen in some cases of heart base tumors. Metastatic lung lesions are common in dogs with hemangiosarcoma.

Advanced imaging techniques such as cardiac computed tomography (CT) and magnetic resonance imaging (MRI) are sometimes used to further characterize structures involving or near the heart. These may be most useful in helping define the extent and location of a mass lesion; however, their ability to diagnose the presence of a cardiac mass has not yet been shown superior to echocardiography.²⁴

Echocardiography

Because echocardiography is highly sensitive for detecting even small-volume pericardial effusion, it is the diagnostic test of choice.^{3,13,25,26} The effusion appears as an echo-free space between the bright parietal pericardium and the epicardium. Abnormal cardiac wall motion and chamber shape and intrapericardial or intracardiac mass lesions can also be visualized.^{22,25-28} Identification of the parietal pericardium in relation to the echo-free fluid helps differentiate pleural from pericardial effusion. Evidence of collapsed lung lobes or pleural folds can often be seen within pleural effusion.

Cardiac tamponade is characterized by diastolic (and early systolic) compression or collapse of the right atrium and sometimes right ventricle (Figure 45-1).^{3,13,27} The left ventricular chamber often appears small, with walls that look hypertrophied (pseudohypertrophy), because of the poor cardiac filling.

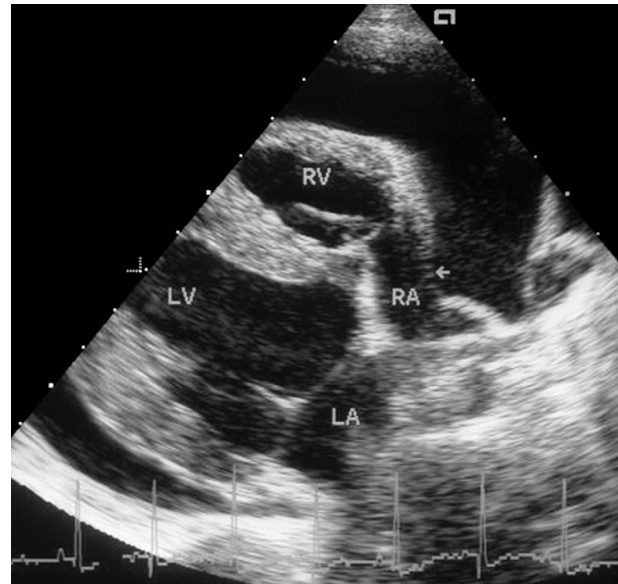


FIGURE 45-1 Right parasternal four-chamber echocardiographic image from a dog with cardiac tamponade. Pericardial fluid is seen surrounding the heart. Note the characteristic collapse of the right atrial wall (arrow) caused by elevated intrapericardial pressure. Electrocardiographic tracing along the bottom. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

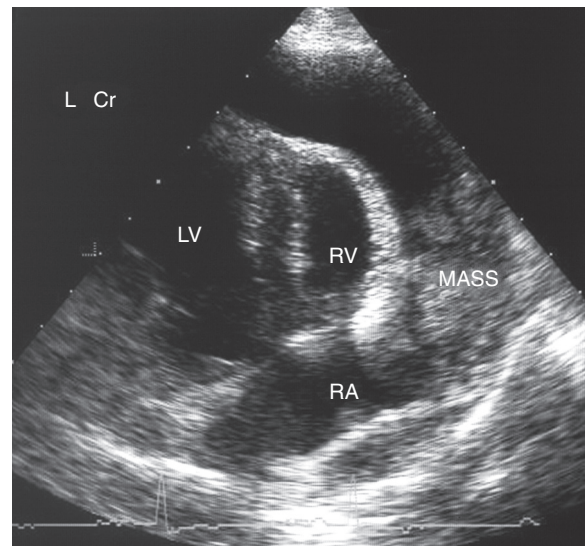


FIGURE 45-2 Left cranial long-axis echocardiographic image, optimized for right ventricular inflow tract and right auricle, from a dog with cardiac hemangiosarcoma. Pericardial effusion enhanced visualization of the tumor (MASS). This echo view confirmed the presence of the tumor and its origin from the tip of the right auricle. LV, Left ventricle; RA, right atrium; RV, right ventricle.

Visualization of structures at the heart base and mass lesions is usually better before pericardiocentesis is performed. It is important to carefully evaluate all portions of the heart, (especially the right atrium and auricle and right ventricle), ascending aorta, and the pericardium itself to screen for neoplasia. The left cranial parasternal (and transesophageal) transducer positions are especially useful (Figure 45-2). Some mass lesions are difficult to visualize. Mesothelioma may not cause discrete mass lesions and therefore may be indistinguishable from idiopathic pericardial effusion.



FIGURE 45-3 Electrocardiogram showing sinus rhythm with electrical alternans, from a dog with large-volume pericardial effusion. Note the every-other-beat change in QRS complex size and configuration in each lead. See text for further information. Leads I, II, III: 50 mm/sec, 1 cm = 1 mV.

Electrocardiography

Although not specific for tamponade, ECG findings associated with large-volume pericardial effusion include reduced amplitude QRS complexes (less than 1 mV in dogs) and electrical alternans. The latter is a recurring, beat-to-beat alteration in the size or configuration of the QRS complex (and sometimes T wave) that results from the heart swinging back and forth within the pericardium (Figure 45-3).²⁹ Electrical alternans may be more evident at heart rates between 90 and 140 beats/min or in certain body positions (e.g., standing). ST segment elevation, suggesting an epicardial injury current, also is seen in some cases of pericardial effusion.^{19,29} Sinus tachycardia is common with cardiac tamponade; atrial and ventricular tachyarrhythmias occur in some cases.

Central Venous Pressure

Central venous pressure (CVP) measurement may be useful in identifying tamponade, especially if it is difficult to assess jugular veins or it is unclear whether right-sided heart filling pressure is elevated. Normal CVP is in the range of 0 to 8 cm H₂O. Cardiac tamponade commonly produces CVP measurements of 10 to 12 cm H₂O or higher.³

Clinicopathologic Findings

Routine laboratory findings may reflect underlying disease or tamponade-induced prerenal azotemia or hepatic congestion but are often otherwise nonspecific.^{3,26} HSA may be associated with a regenerative anemia, increased number of nucleated red blood cells and schistocytes (with or without acanthocytes), leukocytosis, and thrombocytopenia. Pleural and peritoneal effusions associated with cardiac tamponade are usually modified transudates. Circulating cardiac troponin (cTn) concentrations or enzyme activities may be increased as a result of ischemia or myocardial invasion. Elevated

plasma cTnI concentrations have been documented in dogs with cardiac HSA.³⁰

Pericardial Fluid Analysis

Pericardial effusion samples (see [Pericardiocentesis](#) section later in this chapter) should be submitted for cytologic analysis and saved for possible bacterial (or fungal) culture. Nevertheless, differentiation of neoplastic effusions from benign hemorrhagic pericarditis is usually not possible on the basis of cytology alone.³¹ Reactive mesothelial cells within the effusion may closely resemble neoplastic cells; furthermore, chemodectomas and HSAs may not shed cells into the effusion. Effusions associated with lymphoma typically are consistent with a modified transudate, and neoplastic cells usually are easily identified. Many neoplastic (and other noninflammatory) effusions have a pH of 7.0 or greater, whereas inflammatory effusions generally have lower pH. However, there is too much overlap for pericardial effusion pH to be a reliable discriminator.³² Pericardial fluid culture is performed if cytology and pH suggest an infectious or inflammatory cause. It is currently unclear whether analysis of pericardial fluid for cardiac troponins or other substances will allow better differentiation of the underlying etiology.

MANAGEMENT OF CARDIAC TAMPONADE

It is important to differentiate cardiac tamponade from other diseases that cause right-sided congestive signs because its management is unique. The compressed ventricles require high venous pressure to fill. By reducing cardiac filling pressure, diuretics and vasodilators further decrease cardiac output and exacerbate hypotension. Positive inotropic drugs do not improve cardiac output or ameliorate the signs of tamponade because the underlying pathophysiology is impaired cardiac filling, not poor contractility.

Immediate pericardiocentesis is indicated for cardiac tamponade. This sometimes also provides diagnostic information. Congestive signs should resolve after intrapericardial pressure is reduced by fluid removal. A modest dose of diuretic can be given after pericardiocentesis, but this is not essential. Subsequent management is guided by the underlying cause of the pericardial effusion and other clinical circumstances.

PERICARDIOCENTESIS

Preparation and Positioning

Pericardiocentesis is a relatively safe procedure when performed carefully. Depending on the clinical status and temperament of the animal, sedation may be helpful. ECG monitoring is recommended during the procedure; needle or catheter contact with the heart commonly induces ventricular arrhythmias. Although cardiac tamponade is uncommonly caused by coagulopathy, verifying that coagulation parameters are normal is helpful, if patient status allows time for this. Pericardiocentesis usually is performed from the right side of the chest. This minimizes the risk of trauma to the lung (via the cardiac notch) and major coronary vessels, most of which are located on the left. The patient usually is placed in left lateral recumbency to allow more stable restraint; sometimes sternal recumbency is used if the dog is cooperative. Alternatively, the author has had good success using an elevated echocardiography table with a large cutout; the animal is placed in right lateral recumbency and the tap is performed from underneath (Figure 45-4). The advantage of this method is that gravity draws fluid down toward the collection site. But if adequate space is not available for wide sterile skin preparation or for needle or catheter manipulation, this approach is not advised. Echocardiographic guidance can be used but is not necessary unless the effusion is of very small volume or appears compartmentalized. Sometimes pericardiocentesis can be performed successfully on the standing animal, but the risk of injury is increased if the patient moves suddenly.

Several methods can be used for pericardiocentesis. An over-the-needle catheter system (e.g., 16 to 18 gauge, 1.5 inch to 2 inches long) can be used for most cases. Larger over-the-needle catheter systems (e.g., 12 to 14 gauge, 4 to 6 inches) allow for faster fluid removal in large dogs; a few extra small side holes can be cut (smoothly) near the tip of the catheter to facilitate flow, but care should be taken that the end of the catheter does not break off inside the patient. During



FIGURE 45-4 Alternative position for pericardiocentesis using an elevated table with side cutout designed for echocardiography (see text). The animal lies in right lateral recumbency and the puncture is performed from the dependent (right) side.

initial catheter placement the extension tubing is attached to the needle stylet; after the catheter is advanced into the pericardial space and the stylet removed, the extension tubing is attached directly to the catheter. In emergency situations or when an over-the-needle catheter is unavailable, an appropriately long hypodermic or spinal needle attached to extension tubing is adequate. A butterfly needle (18 to 21 gauge) is generally used in cats. For all methods a three-way stopcock is placed between the extension tubing and a collection syringe.

Pericardiocentesis Procedure

The skin is shaved and surgically prepared over the right precordium, from about the third to seventh intercostal spaces and from sternum to costochondral junction. Sterile gloves and aseptic technique should be used. The puncture site is identified by palpating for the cardiac impulse (usually between the fourth and sixth ribs just lateral to the sternum); the optimal site must be estimated if no precordial impulse is felt. Local anesthesia is recommended and is essential with use of a larger catheter. Two percent lidocaine is infiltrated (with sterile technique) at the skin puncture site, underlying intercostal muscle, and into the pleura. A small stab incision is made in the skin when using a larger catheter system.

The puncture site should be just cranial to a rib to avoid the intercostal vessels located caudal to each rib. Once the needle has penetrated the skin, an assistant should apply gentle negative pressure to the attached syringe, three-way stopcock (turned “off” to air), and extension tubing assembly as the operator slowly advances the needle toward the heart. In this way, any fluid will be detected as soon as it is encountered. Pleural fluid (usually straw colored) may enter the tubing first. It is helpful to aim the needle tip toward the patient’s opposite shoulder. The pericardium causes increased resistance to needle advancement and may produce a subtle scratching sensation when contacted. The needle is advanced with gentle pressure through the pericardium; a loss of resistance may be noted with needle penetration, and pericardial fluid (usually dark red) will appear in the tubing. With a catheter system, the needle-catheter unit must be advanced far enough into the pericardial space that the catheter is not deflected by the pericardium as the needle stylet is removed. After the catheter is advanced into the pericardial space and the stylet removed, the extension tubing is attached to the catheter. Initial pericardial fluid samples are saved in sterile ethylenediaminetetraacetic acid (EDTA) and clot tubes for evaluation; then as much fluid as possible is drained.

A scratching or tapping sensation usually is felt if the needle or catheter contacts the heart; also, the device may move with the heart-beat, and ventricular premature complexes are often provoked. If this occurs the needle or catheter should be retracted slightly to avoid cardiac trauma. Care should be taken to minimize extraneous needle movement within the chest. If it is unclear whether pericardial fluid or intracardiac blood (from cardiac penetration) is being aspirated, a few drops can be placed on the table or into a clot tube and a sample spun in a hematocrit tube. Pericardial fluid does not clot (unless associated with very recent hemorrhage). The packed cell volume is usually lower than that of peripheral blood, and the supernatant appears yellow-tinged (xanthochromic). Furthermore, as pericardial fluid is drained, the patient’s ECG complexes usually increase in amplitude, tachycardia diminishes, and the animal often breathes more deeply and appears more comfortable.

Complications of Pericardiocentesis

Ventricular premature beats occur commonly from direct myocardial injury or puncture. These are usually self-limited, resolving when the needle is withdrawn. Coronary artery laceration with myocardial infarction or further bleeding into the pericardial space can occur

but is uncommon, especially when pericardiocentesis is done from the right side. Lung laceration causing pneumothorax or hemorrhage or both is also a potential complication during the procedure. In some cases, dissemination of infection or neoplastic cells into the pleural space may result.

ANCILLARY TREATMENT

Idiopathic Pericardial Effusion

Dogs with idiopathic pericardial effusion are initially treated conservatively by pericardiocentesis. After excluding an infectious cause by pericardial fluid culture or cytologic analysis, a glucocorticoid is often used (e.g., oral prednisone, 1 mg/kg/day, tapered over 2 to 4 weeks); however, its efficacy in preventing recurrent idiopathic pericardial effusion is unknown. Sometimes a 1- to 2-week course of a broad-spectrum antibiotic is used concurrently. Periodic radiographic or echocardiographic reevaluation is advised to screen for recurrence. Cardiac tamponade can recur after a variable time span (days to years). Nevertheless, extended survival times are possible in dogs with idiopathic pericardial effusion, even in those requiring more than three pericardiocenteses.^{18,33} However, recurrent effusions can be caused by mesothelioma or other neoplasia, which sometimes becomes evident on repeated echocardiographic examination.^{7,8}

Recurrent effusion that does not respond to repeated pericardiocenteses and antiinflammatory therapy is usually treated by subtotal pericardiectomy.³⁴ Removal of the pericardium ventral to the phrenic nerves allows pericardial fluid drainage to the larger absorptive surface of the pleural space. The less invasive techniques of thoracoscopic partial pericardiectomy or percutaneous balloon pericardiectomy are also used successfully to treat idiopathic and some cases of neoplastic pericardial effusion.³⁵⁻³⁷ Biopsy samples of a mass (if identified) or even resection of a small right auricular mass can be accomplished through thoracoscopy.

Neoplastic Pericardial Effusion

Pericardiocentesis is done as needed to relieve cardiac tamponade. Attempted surgical resection (depending on tumor size and location) or surgical biopsy, and a trial of chemotherapy (based on biopsy or clinicopathologic findings) can be done; or conservative therapy can be pursued until episodes of cardiac tamponade become unmanageable. Surgical resection of HSA is often not possible because of the tumor's size and extent. Small masses involving only the tip of the right auricle have been successfully removed. Use of a pericardial patch graft may allow resection of larger masses.^{38,39} However, this alone rarely results in prolonged long-term survival. Partial pericardiectomy may prevent the recurrence of tamponade.

Chemotherapy has allowed survival times of 4 to 8 months in some dogs with atrial HSA. Survival time in dogs with mesothelioma may be slightly longer than in those with HSA, but the overall prognosis is poor. Current chemotherapeutic recommendations should be consulted.

Heart base tumors (e.g., chemodectoma) tend to be slow growing and locally invasive and have a low metastatic potential. Partial pericardiectomy may prolong survival for years.

Infectious Pericarditis

Infectious pericarditis should be treated aggressively with appropriate antimicrobial drugs, based on culture and susceptibility testing, and pericardiocentesis as needed. Infusion of an appropriate antimicrobial agent directly into the pericardium after pericardiocentesis may be helpful. If a foreign body is suspected or intermittent pericardiocentesis is ineffective, continuous drainage with an indwelling pericardial catheter or surgical debridement should be pursued.

Surgical therapy allows for removal of penetrating foreign bodies, more complete flushing of exudates, and management of pericardial constrictive disease. Even with successful elimination of infection, epicardial and pericardial fibrosis can lead to constrictive pericardial disease.

CONSTRICTIVE PERICARDIAL DISEASE

Constrictive pericardial disease is recognized occasionally in dogs but only rarely in cats. It occurs when scarring and thickening of the visceral or parietal pericardium restrict ventricular diastolic expansion and prevent normal cardiac filling. Usually the entire pericardium is involved symmetrically. In some cases fusion of parietal and visceral pericardial layers obliterates the pericardial space. In others the visceral layer (epicardium) alone is involved. A small amount of pericardial effusion (constrictive-effusive pericarditis) may be present.

Some cases are secondary to recurrent idiopathic hemorrhagic effusion, infectious pericarditis (especially from coccidioidomycosis but potentially also from other fungal or bacterial infections), a pericardial foreign body, tumors, or idiopathic osseous metaplasia or fibrosis of the pericardium.^{3,13}

Compromised filling reduces cardiac output, and compensatory neurohormonal mechanisms cause fluid retention, tachycardia, and vasoconstriction.

Clinical Features

Middle-aged, medium to large breed dogs are most often affected. Males may be at higher risk. Some dogs have a history of pericardial effusion. Clinical signs of right-sided CHF predominate. These signs may develop over weeks to months. Ascites and jugular venous distention are the most consistent clinical findings, as in dogs with cardiac tamponade.

Diagnosis

The diagnosis of constrictive pericardial disease can be challenging. Typical radiographic findings include mild to moderate cardiomegaly, pleural effusion, and caudal vena cava distention. Echocardiographic changes in dogs with constrictive pericardial disease may be subtle; suggestive findings include mid- and late diastolic flattening of the left ventricular free wall, abnormal diastolic septal motion, and other findings secondary to abnormal hemodynamics. The pericardium may appear thickened and intensely echogenic, but differentiating this from normal pericardial echogenicity may be impossible. Mild pericardial effusion, without diastolic right atrial collapse, is seen in some cases. Serologic testing for *Coccidioides* (or other fungal agents) is advisable in endemic regions.

A CVP greater than 15 cm H₂O is common. Intracardiac pressure measurements are most useful diagnostically. Besides high mean atrial and diastolic ventricular pressures, the atrial pressure waveform shows a prominent y descent (during ventricular relaxation) because ventricular filling pressure is low only in early diastole. This is in contrast to cardiac tamponade, wherein the y descent is diminished. Another classic finding with constrictive pericardial disease is an early diastolic dip in ventricular pressure, followed by a mid-diastolic plateau, but this is not seen consistently. An angiogram may appear normal or may show atrial and vena caval enlargement with increased endocardial-pericardial distance.

Treatment

Therapy for constrictive pericardial disease involves surgical pericardiectomy. If the visceral pericardial layer is affected, epicardial stripping is required, which increases the surgical difficulty and associated

complications. Pulmonary thrombosis reportedly is a common and potentially life-threatening postoperative complication. Tachyarrhythmias are another complication.

CONGENITAL PERICARDIAL DISEASE

Peritoneopericardial diaphragmatic hernia (PPDH) is the most common pericardial malformation in dogs and cats. Abnormal embryonic development (probably of the septum transversum) allows persistent communication between the pericardial and peritoneal cavities at the ventral midline. The pleural space is not involved. Abdominal structures can herniate into the pericardial space, which may cause associated clinical signs.

Clinical Features

Most cases are diagnosed during the first several years of life, usually after gastrointestinal or respiratory signs develop. Vomiting, diarrhea, anorexia, weight loss, abdominal pain, cough, dyspnea, and wheezing are common signs. Physical examination findings can include muffled heart sounds on one or both sides of the chest, a weak or displaced cardiac precordial impulse, an “empty” feel on abdominal palpation (with herniation of many organs), and, rarely, signs of cardiac tamponade. However, some animals never develop clinical signs.

Diagnosis

Thoracic radiographs are often diagnostic or highly suggestive of PPDH. An enlarged cardiac silhouette, dorsal tracheal displacement, overlap of the diaphragmatic and caudal heart borders, and abnormal fat or gas densities within the cardiac silhouette are characteristic findings. Echocardiography may confirm the diagnosis when radiographic findings are equivocal. Other imaging techniques can also be used.

Treatment

Therapy involves surgical closure of the peritoneal-pericardial defect after viable abdominal structures are returned to their normal position. The presence of other congenital abnormalities and the animal's clinical signs influence the decision to operate. In uncomplicated cases prognosis is excellent; however, perioperative complications are common and, although often mild, can include death. Older animals without clinical signs may do well without surgery.

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