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Canine Congestive Heart Failure

Profile

Definition

Congestive heart failure (CHF) is a clinical syndrome associated with a cardiac lesion that results in an increase in venous pressure sufficient to cause a build-up of fluid within the lungs and/or abdomen and/or pleural space.

Signalment

Species. Dogs of any age, sex, breed, or activity level can develop CHF.

Breed predilection. Breeds predisposed to degenerative valvular disease (DVD) and dilated cardiomyopathy are most commonly affected.

- DVD is most common in small breed, older dogs. The prevalence is highest in cavalier King Charles spaniels, dachshunds, toy poodles, miniature schnauzers, and Chihuahuas.
- DCM is most commonly seen in older, large breed dogs. The prevalence is greatest in Doberman pinschers, Great Danes, Irish wolfhounds, boxers, and cocker spaniels.

Causes

- CHF is caused by volume overload of one or both ventricles, resulting in an increase in filling pressures sufficient to cause fluid accumulation within the lungs and/or abdomen and/or pleural space. Any cardiac disease that causes volume overload can cause CHF.
- Although there are several documented causes of CHF in the dog, the majority of cases are secondary to either DVD, or less commonly, DCM.
- Most cases of DCM are thought to be hereditary.
- Nutritional deficiencies, including taurine and carnitine, have been implicated in the etiology of DCM in specific breeds (eg, cocker spaniels).

Risk Factors

The only established risk factor for CHF is development of either DVD or DCM.

Pathophysiology

- The basic pathophysiology of CHF is similar regardless of the underlying cause.
- Although signs of pulmonary congestion (cough, shortness of breath) frequently dominate the clinical picture, the primary abnormality is either myocardial systolic dysfunction (in DCM) or valvular incompetence (in DVD), leading initially to reduced cardiac output.
- Reduced cardiac output activates compensatory mechanisms, including the sympathetic nervous system (SNS) and renin-angiotensin-aldosterone (RAAS) system, which initially restore cardiac output to normal or near-normal levels.
- SNS activation:
  - Causes increased heart rate, increased contractility, and peripheral vasoconstriction
  - Excessive activation of the SNS can be associated with tachyarrhythmias and progressive myocardial dysfunction.
- RAAS activation:
  - Associated with plasma volume expansion from sodium and fluid retention, as well as further vasoconstriction
  - Plasma volume expansion secondary to RAAS activation ultimately leads to elevated venous pressures and edema formation.
- Although initially adaptive, profound and prolonged or chronic activation of these systems is ultimately maladaptive and contributes to disease progression.

Signs

- Common signs of CHF include cough, shortness of breath, exercise intolerance, and syncope.
- Less common signs include abdominal distension, positional respiratory compromise, and isolated nocturnal coughing.
- Patients with DCM, and especially DVD, are frequently identified in the preclinical stage of the disease.

Pain Index

CHF is not thought to be associated with significant pain. Patient anxiety, however, may be associated with development of pulmonary edema and respiratory compromise.

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ACE = angiotensin-converting enzyme; CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; RAAS = renin-angiotensin-aldosterone system; SNS = sympathetic nervous system

Diagnosis

Definitive Diagnosis

- A definitive diagnosis of CHF is established based upon supportive historical findings (ie, tachypnea, cough, exercise intolerance, etc) and physical examination findings (ie, murmur, gallop rhythm, arrhythmia, jugular venous distension, etc) coupled with high-quality thoracic radiographs that identify both pulmonary edema and a cardiac lesion sufficient to cause CHF.
- Resolution of a radiographic infiltrate with a therapeutic challenge of furosemide is considered by many to be the gold standard for diagnosis of CHF (**Figure 1**.)

Other Diagnostic Testing

- Initial blood analysis may demonstrate evidence of renal or prerenal azotemia, mild elevations in liver enzymes, and electrolyte abnormalities.
- Blood abnormalities may become exaggerated following initiation of furosemide and an ACE inhibitor.
- Echocardiography should be performed to confirm the diagnosis. If the patient is *not stable* on room air, the echocardiogram can be delayed until the patient is stable. An echocardiogram cannot confirm a diagnosis of heart failure; it merely confirms the type and severity of structural heart disease.
- Systemic blood pressure should be evaluated to confirm the absence of hypertension, confirm presence and severity of hypotension, and establish a baseline for future reference.
- An ECG is indicated if an arrhythmia is ausculted or suspected based on history.
- Testing NT-proBNP, a circulating biomarker that is elevated in the serum of dogs with CHF, may be useful in dogs with clinical signs suggestive of heart failure, such as dyspnea. The clear clinical utility of this test awaits the outcome of ongoing clinical trials. Although currently published data suggest that values > 300 pmol/L are supportive of CHF, recent reports argue that a more appropriate value might be > 800 pmol/L.
- Other ancillary tests (eg, taurine concentration, *Trypanosoma cruzi* titer, troponin I) may sometimes be useful in dogs with suspected DCM.

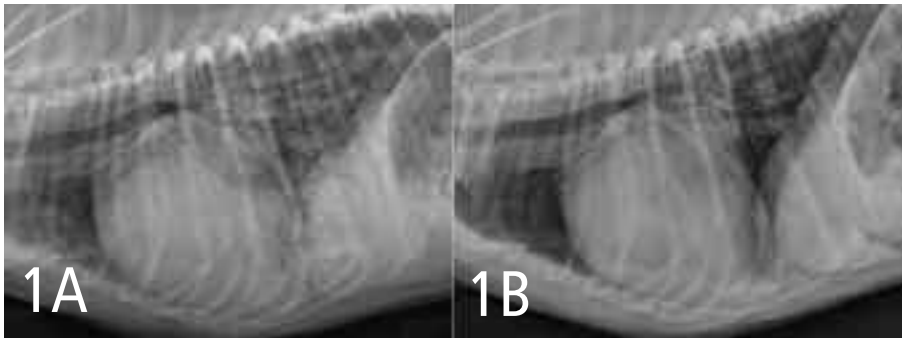
Differential Diagnosis

- Common differentials for cough and shortness of breath include acute primary respiratory diseases, such as infectious bronchitis (kennel cough), chronic small airway disease (chronic bronchitis), dynamic large airway collapse, pneumonia, pulmonary neoplasia, heartworm disease, and pleural/chylous pleural effusion.
- Differentials for collapse and exercise intolerance include diseases of the cardiovascular system in the absence of heart failure (sick sinus syndrome, atrioventricular block [AV block]) the musculoskeletal system (chronic degenerative joint disease, myasthenia gravis), nervous system (intracranial disease, spinal cord diseases), hematopoietic system (anemia), and metabolic derangements (hepatic encephalopathy).

Laboratory Findings/Imaging

Clinicians should be alert to the fact that in presence of cough or signs of respiratory distress, CHF should always be considered a differential diagnosis. Even if thoracic auscultation fails to reveal abnormal lung sounds, a thoracic radiograph should be performed because thoracic auscultation is not highly sensitive for detecting pulmonary edema.

- Radiographically, cardiogenic pulmonary edema in the dog is typically a symmetric, mixed interstitial pattern, worse in the caudal dorsal or perihilar region.
- Other patterns do not rule out cardiogenic pulmonary edema as a differential.
- Definitive diagnosis can be established by documentation of substantial radiographic cardiomegaly or important structural heart disease with an echocardiogram (**Figure 2**), coupled with radiographic clearing of the infiltrate following administration of furosemide (2 to 5 days).
- Resolution of clinical signs is not sufficient to confirm a diagnosis.
- Documentation of structural heart disease with an echocardiogram does not confirm the diagnosis of heart failure, it just means it is possible. Remember that many dogs have preclinical DCM and DVD and could have clinical signs related to other common noncardiogenic diseases (see **Differential Diagnosis**).



Thoracic radiographs from a dog with CHF secondary to DVD before (A) and after 3 days of furosemide therapy (B). Notice the resolution of the pulmonary parenchymal infiltrate but the persistence of the radiographic cardiomegaly.

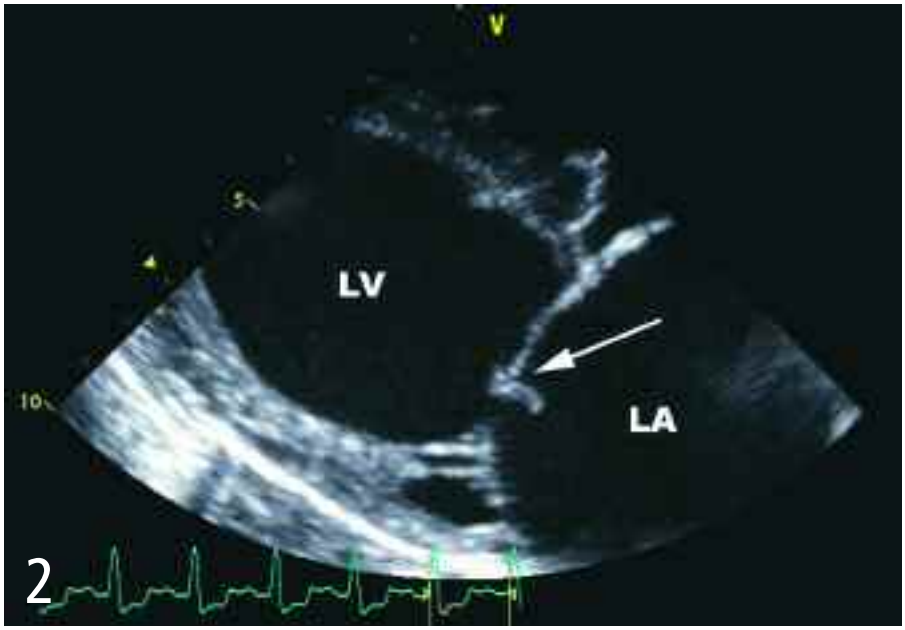
Postmortem Findings

- Gross postmortem findings of CHF are usually those commensurate with pulmonary edema, ascites, and sometimes pleural effusion.
- Generalized cardiomegaly is found, particularly in the left atrium and ventricle (**Figure 3**).
- The mitral valves are thickened, knobby, and sometimes irregular in the case of DVD (**Figure 3**), but typically normal in DCM.

Treatment

Inpatient or Outpatient

- Treatment focuses on the resolution of clinical signs and prolongation of life.
- Resolution of clinical signs of excess plasma volume with appropriate diuresis typically involves the use of furosemide and intermittent abdominocentesis as required, and an ACE inhibitor.
- Improvement and/or preservation of tissue perfusion may include a combination of inotropic support and afterload reduction. If myocardial failure exists, positive inotropic support may increase forward cardiac output and possibly decrease venous filling pressures. If DVD exists, afterload reduction may improve forward cardiac output and decrease volumetric valvular regurgitation. Pimobendan appears to be very effective for both of these applications. Antiarrhythmics are used as required for hemodynamically significant arrhythmias, which are uncommon in DVD but relatively common in DCM.
- Chronic heart failure
  - Conventional therapy for chronic heart failure due to both DCM and DVD includes oral furosemide as needed (1–4 mg/kg Q 8–12 H), an oral ACE inhibitor (enalapril or benazepril) at the label dose, and oral pimobendan (Vetmedin, Boehringer Ingelheim, www.vetmedin.co.uk; 0.25–0.3 mg/kg Q 12 H).
- Acute or decompensated heart failure
  - Management of acute decompensated heart failure can be facilitated by oxygen supplementation and the use of parenteral furosemide (IV or IM) at doses of 1–2 mg/kg Q 1–3 H until the respiration rate has reduced by approximately 50% from baseline.
  - Initiation of oral pimobendan may be beneficial acutely because effects occur within 1 to 3 hours of administration.
  - Addition of an ACE inhibitor can wait until the patient is receiving oral furosemide and is stable.



Right parasternal long axis view from a dog with severe DVD showing a dilated and spherical left atrium and left ventricle. Note the prolapse of the thickened anterior mitral valve *(arrow)* suggestive of chordal rupture. LA = left atrium; LV = left ventricle

Postmortem specimen (whole heart and opened left atrium) from a dog with severe DVD. Note the dilated left atrium and markedly thickened and irregular mitral valve leaflets. Ao= aorta; LA = left atrium

continues

ACE = angiotensin-converting enzyme; AV block = atrioventricular block; CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; NT-proBNP = N-terminal prohormone brain natriuretic peptide; SNS = sympathetic nervous system

- Additional/alternative medications such as spironolactone, digoxin, beta-blockers (atenolol, carvedilol), and hydralazine may be indicated in acute and chronic heart failure management on a case-by-case basis.
- If azotemia develops, cautious IV administration of 1/2 to 1× maintenance low-sodium fluids should be coupled with temporary discontinuation or reduction in diuretic doses.

Contraindications & Precautions

- There are no absolute contraindications when treating a life-threatening disease such as CHF.
- Caution should be used when initiating heart failure therapy in dogs with significant preexisting azotemia or those receiving NSAIDs.

Activity

- Moderate to severe exercise restriction is warranted in patients with severe acute or decompensated CHF.
- Normal activity patterns can be resumed in dogs that become stable on appropriate medical management. Typically, the pet should be allowed to set its own pace.

Client Education

- Clients must be familiar with the clinical signs of impending or overt congestive heart failure, such as cough, shortness of breath, exercise intolerance, and collapse.
- Having clients monitor resting respiratory rate at home may allow them to anticipate impending decompensation, which is frequently preceded by a gradually increasing resting respiratory rate.
- These patients are invariably on diuretics, so it is imperative that they have free access to fresh water at all times.

Nutritional Aspects

- Moderately sodium-restricted diets may be beneficial if tolerated, but diet should be changed only in dogs that are stable on medical management.
- Administration of taurine and carnitine may be of clinical benefit in cocker spaniels with DCM or other nontraditional DCM breeds (any non-Doberman).
- Administration of fish oil has been associated with reductions in some arrhythmias and circulating levels of inflammatory cytokines in dogs with CHF.

Follow-Up

Patient Monitoring

- Once a definitive diagnosis is established, patient follow-up consists of diagnostic tests directed at determining whether CHF is controlled and if complications associated with disease progression and medications are occurring.
- Diagnostic tests typically include a complete medical history to include drug dosing, thoracic radiographs, routine serum biochemical testing, and measurement of systemic arterial blood pressure.

Prevention

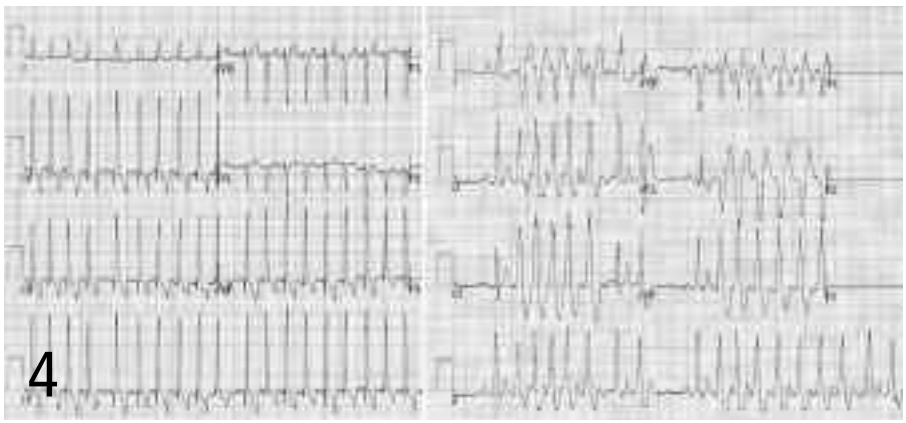
- There are no data to suggest that either of these diseases can be prevented.
- Initiation of therapy in the preclinical stages of DVD is of questionable benefit.
- Identification of DCM in its preclinical stage and initiation of therapy with ACE inhibitors may prolong the preclinical interval.[1]

Complications

- Complications are typically associated with disease progression and heart failure therapy and lead to rapid decompensation.
- Common complications in DCM include development of important arrhythmias such as atrial fibrillation and paroxysmal or sustained ventricular tachycardia (**Figure 4**).
- Complications associated with disease progression in DVD include chordal rupture, left atrial rupture, progressive myocardial dysfunction, and pulmonary hypertension.
- Clinically significant complications associated with standard CHF medications include hypokalemia and azotemia.

Course

- Both these diseases may have a prolonged preclinical stage.
- Once CHF has developed, patients typically have progressively more profound clinical signs that eventually fail to be palliated by medications, or sudden death occurs.



ECGs from a dog with DVD and atrial fibrillation (left) and a dog with DCM complicated by paroxysmal ventricular tachycardia (right).

At-Home Treatment & Monitoring

- Daily administration of multiple medications is necessary for optimal patient management. Having owners monitor resting respiratory rate is helpful. Progressive increases in respiratory rate usually predict clinical decompensation.
- Periodic monitoring of thoracic radiographs, systemic blood pressure, and routine biochemical tests emphasizing renal function and electrolytes help optimize patient care and limit episodes and/or severity of decompensation episodes.

In General

Relative Cost

- Inpatient stabilization of patients with CHF can be costly depending on duration of hospitalization ($$$$–$$$$$).
- Monthly costs associated with outpatient management are relatively modest ($$).

Cost Key

$ = < $100	$$$$ = $500-$1000
$$ = $100-$250	$$$$$ = > $1000
$$$ = $250-$500	

Prognosis

- With very few exceptions, diseases causing CHF in dogs are fatal.
- With appropriate management, median survival times in dogs with CHF secondary to DCM approach 1 year, while dogs with DVD may survive longer than 2 years.
- Good response to short-term/acute management often predicts good long-term survival.
- Many dogs receiving appropriate standard heart failure therapy are completely free of clinical signs and enjoy excellent quality of life for a period of time and clients are typically happy with the outcome of therapy.

See Aids & Resources, back page, for references, contacts, and appendices.

Article archived on www.cliniciansbrief.com.

CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; NSAID = nonsteroidal antiinflammatory drug

sounding board CONTINUED FROM PAGE 9

faucet." Assuming that this use of the paper towel is to prevent clean hands from picking up germs left when the faucet was turned on, isn't it irresponsible to leave germs behind for someone else to pick up? Shouldn't you recommend that the faucet be washed with soap and water before being turned off? Along this same line, I recently read a study that in the human field, stethoscopes are a significant fomite—and probably pens are as well. How many of us sanitize those after working with a possibly contagious animal?

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Editors Respond

Thank you for catching our short-sighted commentary. Of course, we didn't intentionally make recommendations to leave the next person in the bathroom at peril; they were based on use of toilets where you yourself are not the offending person and, as such, do not go far enough. Your remarks are insightful and certainly will benefit readers and give them pause. Thus, we are passing them on.

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