STABILIZATION OF THE CONGESTIVE HEART FAILURE PATIENT IN THE ER

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Emergency and critical care veterinarians commonly care for dogs and cats with congestive heart failure (CHF). Overwhelming, the major keys to success include rest, supplemental oxygen, diuresis and/or thoracocentesis. Other interventions that may be useful include rate control (usually slowing tachycardic hearts, occasionally pacing slow heart rate), positive inotropes, thrombolytics or anticoagulants. Fluids almost never have a role in the care of animals out of heart failure with moderate to severe dehydration. This author’s first choice for hydration in most dogs and cats with active or recent CHF is a “bowl of water”. For this talk, we will consider cats and dogs separately.

CATS

Cardiac disease, common in cats, often leads to congestive heart failure (CHF), arterial thromboembolism (ATE), syncope, or sudden death. Many affected cats show no outward evidence of cardiac disease until one of these major crises has developed. Congenital heart disease can result in clinical signs of heart failure in young cats, although myocardial disease can also be seen in cats less than 1 year of age. Cardiac diseases that more commonly lead to congestive heart failure include hypertrophic, restrictive, and dilated cardiomyopathy as well as uncommon causes such as hyperthyroidism, myocarditis, bacterial endocarditis or other acquired valvular disease. Cats with respiratory distress following administration of fluids (particularly at high rates) and those with dyspnea following anesthesia, or injection of reposital glucocorticoids should be considered as prime candidates to have cardiac disease as a cause of the respiratory distress.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is the most common form of feline heart disease. Affected cats have concentric or asymmetric left ventricular hypertrophy without identifiable cause. Hypertrophic cardiomyopathy can lead to congestive heart failure through a number of mechanisms, often by primarily affecting diastolic function/filling. In some cats, chronic elevation of left atrial pressure can be transmitted back through the pulmonary vascular circuit and can lead to pulmonary hypertension with eventual biventricular heart failure, which is seen as pleural effusion with left atrial enlargement and milder pulmonary edema. Physical exam findings can include increased respiratory rate and effort, loud pulmonary crackles (pulmonary edema), lung sounds may be muffled ventrally (pleural effusion), and jugular vein distension or hepatomegaly (esp. in cats with pleural effusion). Arterial pulses may be normal or weak, mucus membrane color may be normal or somewhat cyanotic, and capillary refill time may be delayed. A prominent cardiac apex beat is often noted on the left side of the chest. Hypothermia is common in cats with CHF and low cardiac output.

In the emergency setting, the most useful diagnostic testing includes history, physical examination, thoracic radiographs and ultrasound. NT-proBNP plasma levels can also be useful to discriminate between cardiac and non-cardiac causes of respiratory distress. Cardiomegaly is usually identified on thoracic radiographs but the concentric nature of the hypertrophy can result in unimpressive cardiomegaly in some cases. Most cats with significant left atrial enlargement will also have enlargement of both pulmonary artery and vein on the DV view. On the lateral view cats with chronic left atrial enlargement often have a tortuous pulmonary vein returning to the left atrium from the caudal lung lobes. Pulmonary edema can be initially manifest as an increased interstitial pattern in the lungs that coalesces into an alveolar pattern as CHF worsens. In many cats this radiographic pattern develops ventrally or is distributed into multifocal, patchy areas of edema rather than in the classic perihilar regions. Pleural effusion can be seen in association with hepatomegaly and enlargement of the caudal vena cava.

Emergency treatment is directed toward management of congestive heart failure, while efforts to reduce the heart rate with beta-blockers or calcium channel blockers are best initiated once clinical signs of CHF are significantly improved. Emergent management of congestive heart failure is often comprised of oxygen, limitation of stress, furosemide (1-4 mg/kg IM or IV q 2-6 hours until better), and perhaps nitrates. It is unclear the efficacy of topical nitroglycerin in the poorly perfused cat. Nitroprusside can be useful in cats that do not respond to furosemide. Sodium nitroprusside is given as a continuous rate infusion, usually starting at 1-2 mcg/kg/minute. The fluid volume should be approximately 1/8th to a total of _ of the cat’s calculated maintenance fluid needs, or
even a smaller volume in cats that are obese. Sodium nitroprusside needs to be protected from light and the drug comes with an opaque bag to shield it from light. Blood pressure measurement is highly desirable for cats with CHF that are being treated with nitroprusside. Unfortunately, the Doppler technique is often difficult to perform in cats with low cardiac output, oscillographic techniques are inaccurate and for all intents and purposes it is impossible to place an arterial catheter in a cat with heart failure. It is often reasonable to give nitroprusside as long as femoral pulses are at least weakly palpable.

Thoracentesis should be performed in cases with a moderate or large volume of pleural effusion. A combination of low doses of diazepam and butorphanol can be used for thoracentesis if sedation is required. It is not necessary to get every drop of fluid, and due to the incomplete nature of the feline mediastinum, excessive angst considering the volumes removed from the right and left sides should be avoided. If any doubt exists as to the cause of the effusion, cytological evaluation and bacterial culture should be performed.

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM), now one of the least common forms of heart disease in the cat, can still be associated with taurine deficiency. Virtually all cats with DCM have developed clinical signs of heart disease by the time of presentation. The history should be checked to determine whether the cat has been eating a diet that might be considered either vegetarian or otherwise unbalanced and subject to reduced dietary taurine intake. A key distinguishing feature of most cats with myocardial failure, especially those with DCM, is the presence of a very loud S3 gallop. Many cats also have either a soft murmur or a cardiac arrhythmia. Cats with DCM and CHF often have developed moderate to large volume pleural effusion. The characteristic echo findings of dilation of all 4 cardiac chambers with reduced left ventricular systolic function and thinning of the walls of the ventricles help to establish the definitive diagnosis. Plasma and whole blood measurement of taurine is indicated in order to help anticipate the response to taurine supplementation. The long term outcome is often related to the presence or absence of taurine deficiency as cats with taurine deficiency often respond well, if they can be made to survive for the first 2-3 weeks. Cats with normal taurine concentrations often respond poorly to medical therapy and may only live for a short time (days to months). In addition to oxygen, thoracentesis (when indicated) and furosemide, other therapies that may be helpful in cats include gentle warming and limited stress. Cats should be discharged from the hospital as soon as possible to avoid stress-induced clinical deterioration. Dobutamine can be given as a continuous rate infusion for cats with refractory hypothermia and azotemia, but this drug is often associated with side effects of vomiting, arrhythmias, and possibly seizures. Dobutamine in our hospital is often given to cats in low doses (1-3 mcg/kg/min) and the infusion is often kept to less than 24 hours as side effect frequency seems to
rise after the first 24 hours. Pimobendan may have a role in treatment of cats with dilated cardiomyopathy at a dose of 1.25 mg/cat q 12 hours.

**Restrictive Cardiomyopathy and Unclassified Forms of Cardiomyopathy**

There is a lack of uniform use of the terms restrictive cardiomyopathy, unclassified cardiomyopathy, and intermediate forms of cardiomyopathy. Some authors feel that most of the cats that do not fulfill the classic criteria for HCM or DCM should be classified as restrictive cardiomyopathy, while others reserve this title for cases with known restrictive physiology (via Doppler studies) or those with restrictive physiology and documented endomyocardial fibrosis. From an etiologic, scientific and classification perspective the distinction might be critical, however there are limited studies to document whether cats with one set of echocardiographic or physiologic patterns respond better to certain interventions. It is the author’s opinion that the clinical response in most of these cases is not determined most tightly by the name applied but by the clinical manifestations of the disease such as the presence or absence of CHF, serious arrhythmia or ATE and the size of the left atrium. It is also the author’s opinion that many cats originally documented to have HCM will have their heart disease progress to having a degree of left ventricular dilation with reduced contractile function and thinning of the walls (towards normal thickness), findings normally diagnosed as restrictive or intermediate cardiomyopathy by most echocardiographers. For these reasons, these disorders will be considered as a single entity termed RCM/UCM.

The majority of cats with RCM/UCM live for many years before clinical signs are seen. Some cats that are asymptomatic at rest will have a history of reduced exercise capacity or open mouth breathing following exertion. In cats with congestive heart failure, the clinical signs of increased respiratory rate and effort are often missed by the owners until they are severe, so the initial manifestations of cardiac disease can be lethargy, weakness, reduced interaction with people or housemates, infrequent vomiting and reduced appetite. Overt respiratory distress often follows, usually within 1-3 days, and increased respiratory rate and effort are often magnified on exam due to the stress of transportation to the hospital and the new environment. ATE may be a more common reason for presentation of cats with the endocardial form of restrictive cardiomyopathy. Physical examination findings are not specific for RCM/UCM and can include cardiac murmur, cardiac gallop, or cardiac arrhythmia. Cats with isolated left heart failure and pulmonary edema usually develop loud pulmonary crackles, often with severe respiratory distress. In cats with biventricular heart failure (usually the result of longstanding left heart failure) respiratory distress is often accompanied by jugular vein distention, hepatomegaly, and muffled lung sounds ventrally due to pleural and/or pericardial effusion. A small volume ascites may be present in cats with biventricular heart failure; however marked cardiogenic ascites in the cat is usually due to isolated right heart disease. Cats with CHF are often hypothermic, mucous membrane color may be cyanotic, capillary refill time may be delayed. They often appear to be dehydrated based on skin turgor despite the fact that they clearly have excessive circulating intravascular volume based on the presence of pulmonary edema, pleural effusion, and jugular vein distention. Weak arterial pulses are common once congestive signs develop. Thoracic radiographs are indicated to establish the diagnosis of CHF and to differentiate heart disease from respiratory or heartworm disease. Blood pressure should be routinely measured in cats with heart disease. Cats over 6 years of age should be tested for hyperthyroidism, but one should keep in mind that cats with advanced heart disease can have low T4 levels (below the normal range) due to euthyroid sick syndrome, so thyroid supplementation is usually not appropriate. Echocardiography is the best tool to distinguish RCM/UCM from other forms of feline heart disease.

Therapy of cats with congestive heart failure due to RCM/UCM is similar to that described above for dilated cardiomyopathy. Due to the high incidence of ATE in cats with this form of myocardial disease, especially since volume contraction can result in worsening vascular stasis, the author sometimes employs heparin at 250 units/kg subcutaneously 3 times a day while cats are in the hospital for management of congestive heart failure. Clopidogrel (1/4 of a 75 mg tablet) is another anti-platelet/clot option for growing in popularity for prevention of clots in cats, although like all oral drugs is probably best delayed until CHF has resolved and a normal appetite has returned.

**DOGS**

Acquired heart disease is the most common cause of congestive heart failure in dogs, specifically chronic valvular disease and dilated cardiomyopathy. However, other possible causes of heart disease/heart failure are also occasionally seen such as congenital disease (eg L-R PDA), or endocarditis. Severe heartworm disease and pulmonary hypertension may also result in clinical signs. In this author’s opinion, the incidence of pulmonary hypertension has been rising in recent years, although since right-sided CHF often is not present, the respiratory
distress and pulmonary infiltrates that can be seen in pulmonary hypertension must be distinguished from animals with congestive heart failure.

Emergency treatment is directed toward management of congestive heart failure, control of arrhythmias, centesis of effusions causing marked alterations in cardiopulmonary function and restoration of tissue perfusion. Emergent management of congestive heart failure is often comprised of oxygen, limitation of stress, furosemide, and perhaps nitrates. Thoracentesis should be performed in cases with a moderate or large volume of pleural effusion, and pericardiocentesis is usually indicated in dogs with cardiac tamponade. A combination of low doses of diazepam and a narcotic (in combination with a local block if desired) can be used for thoracentesis if sedation is required (the author recommends against the use of propofol in this setting).

**Furosemide**

Furosemide is probably the most useful drug for the management of acute congestive heart failure and is the diuretic of choice for the management of severe pulmonary edema due to heart failure. In dogs with acute severe pulmonary edema, high doses of furosemide (up to 4 mg/kg IV every 1-2 hours) may be required to induce a diuresis. In many cases, the diuretic causes a significant contraction of intravascular volume at a time when some respiratory difficulty is still present. Continued use of furosemide can lead to azotemia, severe hypotension, electrolyte depletion, and metabolic alkalosis. It is quite difficult to define the exact dose of diuretic required by any individual dog with CHF. The dose required to clear significant edema accumulations and cause the animal to be minimally symptomatic (the desired dose) is often close to a dose that might result in electrolyte disturbance, dehydration and the development of pre-ren al azotemia. The combined use of ACE inhibitors and diuretics compromises one of the kidneys' normal compensatory mechanisms (vasoconstriction of the efferent arteriole) and can lead to elevation of BUN and creatinine when 1) excessive diuretic dose is initiated or 2) significant pre-existing renal disease is present. For this reason, high doses of diuretics that are often needed to control severe pulmonary edema often lead to a rise in BUN or creatinine over the next few days. At the time of hospital discharge the author often provides the owner with upper and lower limits for acceptable furosemide dose. In the emergency setting, an alternative to pulsatile high dose furosemide is to administer furosemide using a continuous rate infusion in a small volume of fluid. The dose is usually adjusted to between 0.1 and 1 mg/kg/hour. This method may be associated with superior diuretic effect and fewer electrolyte derangements although requires an infusion pump and perhaps more intensive nursing care.

**Nitroglycerin**

Nitroglycerin acts as a nitric oxide (NO) donor to create vasodilatation. The venous vasodilatation is thought to account for at least some of the beneficial effect that occurs in acute CHF. Arterial vasodilatation and coronary artery dilation might also contribute to either clinical benefit or side effects. The drug is most often used in the setting of acute severe pulmonary edema, for 1-3 days, until other medications can be initiated. Typical doses of 1/8” to 2” are applied topically (transdermally), using gloves to avoid absorption to the person administering medication, and the drug is given q 6 to 8 hours. There is some controversy as to the effectiveness of this transdermal drug administration in the setting of CHF, and the drug is more often given intravenously for people with CHF. The most common side effects are hypotension, weakness, lethargy, and presumably headache (a common complication in humans with CHF).

**Sodium Nitroprusside**

Nitroprusside can be useful in dogs that do not respond to furosemide. In our hospital, sodium nitroprusside is most commonly initiated in animals with life-threatening pulmonary edema and minimal clinical improvement after 1 to 3 doses of furosemide at 4 mg/kg IV. This drug can be very effective in this setting, although blood pressure monitoring is ideal. Close observation of the animal, skilled technicians, and frequent re-evaluation of the animal’s condition is needed to find an effective dose. Doses ranging between 2 and 10 mcg/kg/min are often successful. The mean arterial pressure should ideally be maintained above 65 mmHg, although even in dogs with severe CHF and concurrent hypotension this drug seems to be tolerated and is useful for short term (8 to 12 hour) management. Sodium nitroprusside is often initiated at 1-2 mcg/kg/minute in a fluid volume equal to approximately 1/16th to 1/8th of the animal’s calculated maintenance fluid needs. Sodium nitroprusside needs to be protected from light and the drug comes with an opaque bag to shield it from light.
Dobutamine

Dobutamine can be given as a continuous rate infusion for dogs and cats with CHF, hypothermia and azotemia. These animals with CHF and low cardiac output often have cold extremities, hypothermia, pallor and slow CRT, azotemia, weakness and decreased blood pressure. Dobutamine has a short half-life and must be given by continuous IV infusion, preferably administered through an infusion pump. In dogs, the infusion rate is adjusted upward from 2.5 mcg/kg/min at 2.5 mcg/kg/min increments until signs of improved cardiac function appear such as increased blood pressure, warm limbs, improved mucous membrane color and CRT, decreased respiratory distress. The typical effective dose is between 5 and 15 mcg/kg/min in dogs. Increases in heart rate greater than 20% above baseline, heart rates above 190 beats/minute or arrhythmia formation dictates dose reduction. Recent retrospective studies from people with acute decompensated congestive heart failure have failed to show clear benefit from dobutamine. Several studies have suggested that use of positive inotropes are associated with adverse outcome or shortened survival, even in the setting of acute CHF and short-term administration of dobutamine or milrinone. Dogs that have been receiving ß blocker therapy are not candidates for dobutamine therapy, or they may require higher doses of inotropes in order to achieve the desired clinical effect, and these higher doses can be associated with arrhythmia.

Pimobendan

Pimobendan is a calcium sensitizing drug that is useful as a positive inotrope in addition to having properties as a phosphodiesterase inhibitor with vasodilating effects. It has been studied in dogs with chronic valvular disease and in dogs with dilated cardiomyopathy. Pimobendan can result in significant clinical improvement in dogs when it has been added to background therapies of furosemide and an ACE inhibitor for heart failure. While pimobendan has mostly been studied in the setting of treatment of chronic congestive heart failure, there is some hope that the drug could be an adjunct to management of emergent CHF as well. The drug is rapidly absorbed from the gastrointestinal tract, usually within 1-2 hours, and this rapid onset of action may prove useful in the emergency setting. The usual dose for pimobendan for treatment of chronic heart failure in dogs is 0.25 mg/kg q 12 hours.

Angiotensin-Converting Enzyme Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are commonly used in the management of chronic CHF and the main benefits of ACE inhibitors are not seen in an emergency setting. However, enalaprilat can be given IV and we have used the injectable formulation of enalaprilat in a number of dogs and cats with severe pulmonary edema.

Mechanical Ventilation

In certain cases, mechanical ventilation is desirable to control the airway, avoid respiratory muscle fatigue, and initiate positive end-expiratory pressure to help mobilize edema while other therapies take effect. Most animals with congestive heart failure are markedly improved in 24 to 48 hours; if improvement is not detected, both the current therapeutic plan and the diagnosis should be reconsidered.

References available upon request