Feline cardiomyopathies

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Hypertrophic cardiomyopathy (HCM) is the most common heart disease in cats. Fifteen to thirty-four percent of healthy cats have echocardiographic evidence of left ventricular concentric hypertrophy (increased ventricular wall thickness) attributed to HCM. HCM is caused by a primary defect within the heart muscle cells, which causes the ventricle to become thick (i.e., concentric hypertrophy) and develop excess scar tissue (i.e., fibrosis), which makes the ventricle stiff and unable to relax normally. HCM is inherited in Maine coon cats and Ragdoll cats as an autosomal dominant trait with incomplete penetrance. This means that HCM affects all individuals that have the mutation, but to varying degrees. HCM ranges in severity from mild to severe disease. Pathophysiologic sequelae to severe HCM may be development of diastolic congestive heart failure (i.e., pulmonary edema and/or pleural effusion), arterial thromboembolism, or sudden death.

The cardinal pathophysiologic characteristic of hypertrophic cardiomyopathy is impaired diastolic filling of the left ventricle, due to abnormal relaxation of the heart muscle and increased ventricular muscle stiffness. Diastole is comprised of active isovolumic relaxation, rapid passive filling, diastasis, and atrial systolic filling. Cats with HCM have abnormal relaxation and increased stiffness that impairs passive ventricular filling. Impaired relaxation is caused by abnormal calcium handling, increased myofilament sensitivity to calcium, intracytosolic calcium overload, altered left ventricular loading conditions, and myocardial ischemia from small coronary artery disease. Increased ventricular stiffness is caused by concentric left ventricular hypertrophy, myofiber disarray, and myocardial fibrosis. Delayed relaxation and increased ventricular stiffness increase diastolic filling pressure, which may lead to development of left heart failure. Pulmonary edema and/or pleural effusion develop as the main manifestations of left-sided congestive heart failure in cats with HCM. Systolic anterior motion (SAM) of the mitral valve develops secondary to anterior-ventrally displaced, hypertrophied papillary muscles that pull the mitral valve into the left ventricular outflow tract during systole.

Other factors that may exacerbate or worsen SAM of the mitral valve include

severe basilar septal concentric hypertrophy, increased contractility, and tachycardia. Moderate or severe SAM of the mitral valve greatly increases left ventricular systolic pressure, which increases severity of concentric LV hypertrophy and potentiating the vicious cycle of hypertrophy and the potential for worsened diastolic function. Arterial thromboembolism may occur in cats with left atrial enlargement. Factors involved in development of a left atrial thrombus include blood stasis, possible endothelial disruption, and a possible procoagulable state with increased platelet aggregation and coagulopathic markers.

Restrictive cardiomyopathy (RCM) is less common that HCM, but also primarily causes diastolic dysfunction. Dilated cardiomyopathy, now a rare cause of heart disease in the cat, is the only cardiomyopathy causes systolic dysfunction (although often there is also diastolic dysfunction). Differentiation of the form of cardiomyopathy requires an echocardiogram, which demonstrates increased left ventricular or interventricular septal end-diastolic wall thickness (with HCM), normal wall thickness with a restrictive filling pattern (with RCM) or thinner than normal wall thickness and reduced contractility (with DCM). With any of the cardiomyopathies left atrial dilation may be present. Congestive heart failure is diagnosed by clinical signs and thoracic radiographic evidence of cardiomegaly, pulmonary edema and/or pleural effusion, and often pulmonary venous distension may also be present with any of them.

Electrocardiography is an insensitive test to screen for HCM, but it is important in cats with an arrhythmia or history of episodic weakness or collapse. Hyperthyroidism, systemic hypertension, subaortic stenosis, and acromegaly are differential diagnoses for increased left ventricular wall thickness seen with HCM. Basic comprehensive blood work including complete blood count, serum chemistry, total thyroxine level, and urinalysis is recommended in cats diagnosed with HCM, especially in middle-aged to older cats, or in cats with heart failure. Baseline renal function is important to assess prior to medical treatment of heart failure. Significant anemia should be identified, as this may significantly worsen ventricular volume overload (i.e., preload) in cats with HCM. Total thyroxine concentration is an essential diagnostic test in middle-aged to older cats with echocardiographic evidence of concentric hypertrophy, because hyperthyroidism is a secondary cause of concentric hypertrophy. Serum growth hormone levels may be mildly increased in cats with HCM, but they are much higher in cats with acromegaly. Insulin-like growth factor-1 $\pm$ serum growth hormone should be measured in cats with concentric hypertrophy of the left ventricle and clinical abnormalities suggestive of

acromegaly.

Treatment of cats with HCM is dependent on many variables, which may include presence of left atrial dilation, severity of systolic anterior motion of the mitral valve, tachycardia, severity of left ventricular hypertrophy, client and patient motivation and ability to chronically administer medications. Treatment options for asymptomatic cats include atenolol, or less preferably diltiazem. If elected, atenolol is the treatment of choice for moderate or severe systolic anterior motion of the mitral valve (left ventricular to aortic pressure gradient measured on echocardiography of **≥**50 mm Hg), in the appropriate clinical context.

There is significant controversy regarding when to initiate treatment, and what is the most appropriate medical therapy in asymptomatic cats with HCM and no heart failure. Beta blockers (i.e., atenolol) or calcium channel blockers (i.e., diltiazem) are the most commonly used drugs in asymptomatic cats with HCM, and they may reduce severity of hypertrophy. Beta blockers are more effective than calcium channel blockers to reduce severity of systolic anterior motion of the mitral valve and control heart rate to prevent tachycardia. Clinical evidence from placebo controlled, blinded clinical studies indicate that early use of angiotensin converting enzyme inhibitors or aldosterone antagonists in cats with asymptomatic HCM and no heart failure is not warranted. Prophylactic anticoagulant therapy is indicated if there is echocardiographic evidence of spontaneous contrast (i.e., red blood cell aggregation) or an intracardiac thrombus. It is controversial whether cats with significant left atrial dilation should be placed on prophylactic anticoagulation. Prophylactic anticoagulant therapy is not necessary in cats with mild HCM and normal left atrial size, except in the rare incidence where such a cat has a history of confirmed ATE.

Congestive heart failure is treated with furosemide and an ACE inhibitor. Anticoagulant therapy with clopidogrel, aspirin, or low molecular weight heparin is necessary in cats currently or previously suffering from arterial thromboembolism, cats with echocardiographic evidence of spontaneous contrast or thrombus, and may be considered in cats with moderate to severe left atrial dilation (left atrial to aortic ratio of **>**1.9 and or evidence of atrial blood stasis). Prognosis of mild and/or asymptomatic HCM is good, and cats may live for many years without problems. The prognosis worsens once cats develop congestive heart failure, with average survival times ranging from 92–654 days. Cats with HCM and ATE have the poorest prognosis, with average survival times ranging from 61–184 days.

Furosemide is the most effective and life-saving treatment of cats in congestive heart failure and can be given at a dose range of 1–2 mg/kg PO q 24 hr to TID for outpatient therapy depending on the severity of heart failure. The minimal effective dose should be used. An initial moderate to high dose may be started and then rapidly tapered based on respiratory rate, effort, and evaluation of severity of heart failure by thoracic radiographs. Treatment of acute heart failure includes parenteral furosemide (1–2 mg/kg q 1–8 hr). The dose and frequency should be rapidly tapered once the respiratory rate decreases to 50 bpm or less and the effort is decreased. Oxygen therapy with 60–70% FiO2 can be done initially and then decreased to 50% or less within 12 hours to avoid further barotrauma secondary to high inspiratory oxygen concentration. Transdermal nitroglycerin causes venodilation in people, but it has not been evaluated in cats. Its use is debatable in cats and can be given short-term to hospitalized cats for no longer than 2 days; the main drawback appears to be a lack of efficacy rather than adverse effects. Negative inotropic therapy including beta blockers and calcium channel blockers may be used in some cats with chronic heart failure but are not given in acute heart failure unless there is a hemodynamically significant tachyarrhythmia. ACE inhibitors such as enalapril or benazepril may help provide adjunctive treatment in cats with heart failure. Rather than starting as an emergency in-hospital treatment, the ACE inhibitor may be started at home once the cat is stabilized, eating and drinking, and hydrated. Prophylactic anticoagulation may be started in cats with high risk of arterial thromboembolism, including those with spontaneous echocardiographic contrast, left atrial thrombus, history of prior ATE, or severe left atrial dilation. Congestive heart failure, arterial thromboembolism, and sudden cardiac death are the most common and devastating clinical sequelae in cats with cardiomyopathy. Monitoring of asymptomatic cats with HCM includes repeat echocardiograms every 4–12 months depending on the severity and progression of the disease. To evaluate for early heart failure, radiographs should be obtained periodically (every 3–6 months) in cats with significant left atrial dilation. Monitoring of cats with HCM and heart failure includes repeat thoracic radiographs and renal panels to assess clinical response and guide medical therapy. Thoracic ultrasound can be done to evaluate severity of pleural effusion and guide location for thoracocentesis. Systolic blood pressure and thyroxine levels should be periodically evaluated in middle-aged to older cats that are at risk for developing systemic hypertension or hyperthyroidism.