VETERINARY SPECIALTY CENTER
EMPLOYEE-OWNED

2051 Waukegan Road, Bannockburn, IL 60015
847.459.7535 Fax 847.808.8900
Email: cardiology@vetspecialty.com
vetspecialty.com

Hypertrophic Cardiomyopathy (HCM)

Background

Hypertrophic cardiomyopathy (HCM) is a general diagnosis provided for when the muscular walls of the ventricles (lower heart chambers) become abnormally thickened (hypertrophied). There are several etiologies that can result in HCM. The most common cause is a primary heart muscle disease called idiopathic HCM. Idiopathic HCM is genetic in many cases based on both veterinary and human studies and the prevalence of the disease in certain breeds (Maine Coons, Ragdolls, Bengals, Sphinx, etc.). Other less common causes of HCM include elevated blood pressure (i.e. systemic hypertension), mitral valve dysplasia causing an outflow tract obstruction, hyperthyroidism (elevated thyroid levels), infiltrative disease (i.e. cancer, amyloidosis), and transient thickening due to inflammation (rare). Idiopathic HCM is diagnosed when these other causes have been ruled out. The overall prevalence of idiopathic HCM is approximately 15%, making it relatively common in the general population and the most common acquired heart disease of cats.

The hallmark problem with HCM is the inability of the left ventricle to relax appropriately due to the thickening and stiffness of the left ventricular walls. In a subset of cats, the thickening becomes substantial enough to cause an increase in pressure within the heart. Eventually, this increased pressure may build in the lungs and can result in leakage of fluid from the blood vessels within the lungs (called pulmonary edema) or around the lungs (called pleural effusion). When this occurs, it is referred to as congestive heart failure (CHF). Pulmonary edema and pleural effusion make breathing difficult since the lungs can't exchange oxygen and other gases appropriately. Clinical signs associated with CHF include lethargy/weakness, decreased activity level, and an increase in breathing rates/effort. If any of these signs are noted, a veterinarian should be contacted immediately.

Other common secondary results to HCM include aortic thromboembolism (ATE), arrhythmia, and outflow tract obstruction.

- When the top chamber of the heart (atrium) on the left side begins to dilate as a result of HCM, blood clots may form. When these blood clots leave the heart and lodge in the aorta, they are referred to as ATE. Most commonly, ATE causes blockage of blood flow to the back legs resulting in sudden paralysis and severe pain. Depending on the size of the ATE, some animals recover spontaneously, while others never regain full use of their extremities or die from complications of the clot. A medication called clopidogrel is often prescribed to mitigate the risk of clot formation.
- Arrhythmia, including atrial fibrillation and ventricular tachycardia, can occur due to structural damage to the heart as it enlarges. Depending on the severity, anti-arrhythmic medications may be recommended. Regardless, these abnormal heartbeats can predispose cats to sudden death.
- Obstruction to blood flow leaving the left side of the heart may occur due to thickening of the heart muscle within the outflow tract (i.e. just below the aortic valve) or from the abnormal movement of the mitral valve (valve separating the top and bottom chambers on the left side of the heart) due to alteration of the valve apparatus from the thickening heart muscle. This outflow tract obstruction increases the workload on the heart muscle, resulting in a more rapid progression of thickening. Depending on the severity, a medication called atenolol may be prescribed in an effort to resolve or mitigate the severity of the obstruction.

The diagnostic work-up for HCM often includes an echocardiogram, chest x-rays, ECG, blood pressure assessment, and blood work including a kidney panel and a thyroid panel. Specific diagnostics tests are chosen based on history as not all of these tests are necessary in every patient. Occasionally, a BNP test (B-type natriuretic peptide) is recommended as a screening or diagnostic tool.

Treatment

There is no cure for idiopathic HCM. As of late 2025, a conditionally FDA approved medication, Felycin-CA1, became available that may slow the thickening associated with some forms of HCM. No long-term safety/efficacy studies are available, however, and it is unknown at this time if the medication improves survival times. It is contraindicated in cats with diabetes and liver disease, and the safety has not been evaluated in cats with kidney disease, hyperthyroidism, chronic viral infections, or in those receiving steroids or beta-blocker therapy (ex: atenolol). Beta-blockers may increase the risk of toxicity. It has NOT been approved for usage in patients with a history of CHF.

For these reasons, this medication is only being considered for select cases. Medications such as diuretics, ACE inhibitors, and pimobendan/Vetmedin may be prescribed to treat signs associated with CHF and slow the accumulation of fluid in and/or around the lungs. Additionally, medications to reduce the risk of clot formation (ex: clopidogrel, rivaroxaban), outflow obstruction (ex: atenolol), and/or arrhythmia are sometimes recommended.

Prognosis

The prognosis for cats with HCM is highly variable with the majority of patients never progressing to the stage of CHF. Only 7% of cats with stage B1 HCM (normal to mild left atrial dilation) die of their disease within 1 year of diagnosis. The 5- and 10-year cardiac mortality rates are 23% and 28%, respectively, meaning that many cats stay in the subclinical stage for years, and often for life. Once the stage of CHF has been reached, the median survival time is 9-12 months and highly dependent on comorbidities (ex: concurrent kidney disease, clot formation) and the individual's response to medications.

Since the disease process continues regardless of treatment, those patients who progress to CHF will often have multiple episodes. The severity of subsequent episodes can often be mitigated with close monitoring for clinical signs of CHF, especially increases in sleeping respiratory rates. At each episode, the medications are often increased or new medications may be added to further decrease the workload on the heart. Between episodes of CHF, the vast majority of patients experience an excellent, near-normal quality of life.