

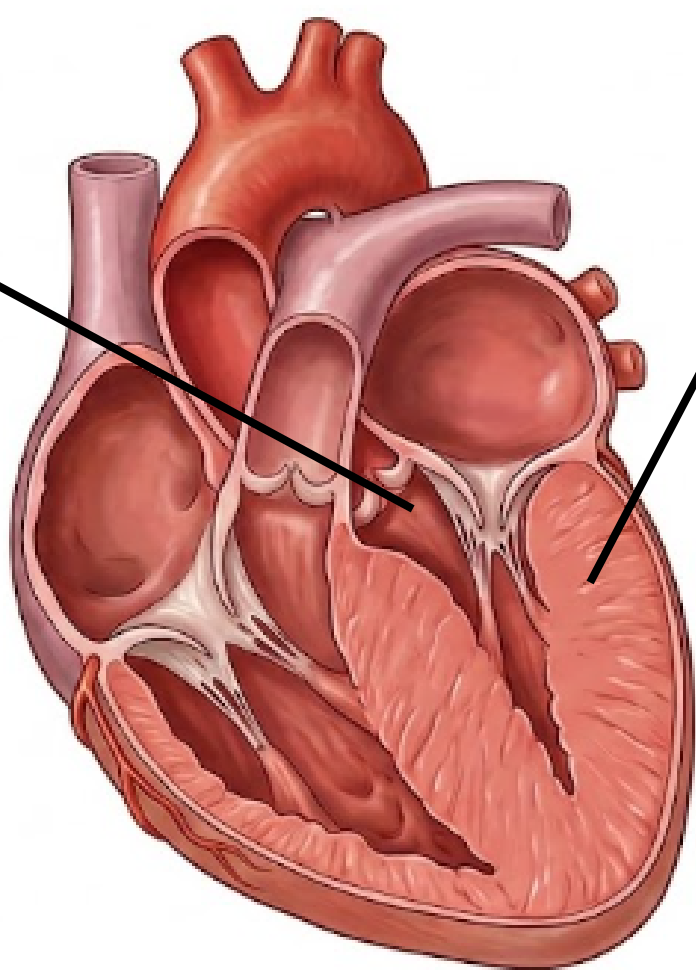
EARLY DIAGNOSIS OF FELINE HYPERTROPHIC CARDIOMYOPATHY

OBJECTIVES

The aim of this review is to compile and analyze current scientific evidence to provide an overview of this pathology, emphasizing the available tools for early diagnosis.

Hypertrophic cardiomyopathy (HCM) is a myocardial disorder that involves diffuse or regional thickening of the left ventricular (LV) wall without concurrent left ventricular dilatation. It has a prevalence of approximately 15% in cats.

DLVOTO and SAM: High-velocity systolic blood flow drags the mitral valve leaflet into the outflow tract, causing a dynamic obstruction and mitral regurgitation. This increases LV workload accelerating left atrial dilatation, and increasing the risk of congestive heart failure and arterial thromboembolism.



Diastolic dysfunction: Myocardial fibrosis and LV hypertrophy reduce ventricular compliance. This results in elevated filling pressures that transmit retrogradely, causing left atrial dilatation, and in severe cases, leading to pulmonary edema or pleural effusion.

Predisposed breeds

Maine Coon (A31P mutation), Ragdoll (R820W mutation), Sphynx, Bengal, British Shorthair, Persian, Siberian, Norwegian Forest Cat, and others.

Main clinical signs

Tachycardia, tachypnea, exercise intolerance, dyspnea, heart murmur, arrhythmias, cyanosis and cold extremities.

Diagnostic Tool	Description	Advantages	Limitations
Genetic Testing	<i>MYBPC3</i> mutation: A31P in Maine Coon and R820W in Ragdoll.	Exclusion of affected individuals from breeding programs.	Breed-specific. Negative result does not rule out the disease.
NT-proBNP	Response to myocardial stretch, measured via quantitative or rapid POC tests.	Differentiates cardiac and non-cardiac causes of dyspnea.	Cannot differentiate types of cardiomyopathies or the stage.
Cardiac Troponin I	Indicates damage to cardiomyocytes.	Highly elevated levels are associated with risk of cardiovascular death.	Other cardiac or systemic diseases can elevate its levels.
Galectin-3	Reflects tissue inflammation and myocardial fibrosis.	Shows potential for identifying advanced stages of HCM.	Current feline clinical studies show conflicting results.
Thorax X-ray	Imaging technique, requiring at least two views.	Confirms pulmonary edema and pleural effusion in advanced stages.	Cannot differentiate between cardiomyopathy types.
Electrocardiography	Evaluates the heart's electrical activity.	Tpeak-Tend interval shows potential to correlate with disease severity.	Different cardiac abnormalities can produce similar electrocardiographic patterns.
Echocardiography	Assesses cardiac structure, function and hemodynamics.	Confirms the hypertrophic phenotype (LV wall >6 mm), and enables disease staging.	Specialized equipment and training. Cannot differentiate primary from secondary hypertrophy without ruling out systemic causes.
Speckle-Tracking Echocardiography	Advanced imaging software that quantifies myocardial strain and strain rate.	Studies suggest high sensitivity for detecting early subclinical myocardial dysfunction.	Still needs further research to establish standardized feline reference ranges.

CONCLUSIONS

Echocardiography remains the most effective diagnostic tool. Combining it with other techniques would be highly valuable for early disease detection. However, further studies evaluating the functionality of early-detection tests are essential to integrate them into daily clinical practice.