

# Feline Hypertrophic Cardiomyopathy: An Update

*K.M. Meurs*

## Section snippets

### Introduction/Historical Perspective

Recently proposed classifications of human cardiomyopathies have emphasized the etiology or molecular basis of myocardial disease.<sup>1, 2</sup> Although the cause of a few specific breed-associated feline cardiomyopathies has been determined, feline myocardial disease remains largely idiopathic.<sup>3, 4</sup> Accordingly, morphopathologic/functional designations remain valid. Given that premise, cardiomyopathy can be defined as a heart muscle disease that is associated with dysfunction.<sup>5</sup>

Hypertrophic

### Etiopathogenesis

It is now established that HCM in human beings is a genetic disease.<sup>8, 17</sup> Several hundred distinct genetic mutations have been associated with HCM; genetic testing together with pedigree analyses have demonstrated that these mutations are casually related to the HCM phenotype.<sup>18, 19, 20</sup> Although exceptions have recently been identified, causative mutations primarily affect genes that encode proteins that are incorporated into the contractile elements, or sarcomeres, of the myocyte.<sup>21</sup>

Among

### Epidemiology

Population characteristics of feline HCM have been retrospectively evaluated.<sup>29, 30</sup> A sex predisposition for males is consistent and the mean age at diagnosis is close to 6 years.<sup>29, 30, 31</sup> Despite the fact that these data were obtained from referral populations, a substantive proportion, between 33% and 55%, were subclinical (asymptomatic) when the disease was identified.<sup>29, 30, 31</sup> One of these investigations identified the administration of corticosteroids as a historical antecedent to the

### Diastolic Dysfunction

It has generally been accepted that diastolic dysfunction is the pathophysiologic mechanism that is primarily responsible for the clinical manifestations of HCM. That systolic function contributes

prominently to cardiac performance is readily evident but the importance of diastolic function is less obvious. Diastolic function (the ability of the ventricle to fill at low pressure) is complex but depends on the energy-dependent process of myocardial relaxation and mechanical properties of the

## **Clinical presentation**

Feline HCM is usually identified when auscultatory findings, such as arrhythmias, gallop sounds, or murmurs, are incidentally detected during routine veterinary examinations or when clinical signs result from heart failure or embolism.<sup>29, 30</sup> In a few affected cats, sudden unexpected death is the first clinical manifestation of the disease. Respiratory distress related to pulmonary edema or sometimes, pleural effusion, is the most common clinical manifestation of heart failure in feline HCM.

## **Screening for HCM**

Recent interest in the epidemiology of feline heart disease raises questions regarding screening for this disease. The cost-benefit ratio of screening for clinically occult disease is favorable if an affordable test can identify a disease that is serious and treatable. Feline HCM, in its severe form, is undoubtedly serious and is clearly an important cause of morbidity and mortality in cats. Unfortunately, there is little known of the natural history of feline HCM; the rate at which subclinical

## **Subclinical HCM**

Optimally, it would be possible to identify patients that have a subclinical form of HCM that is destined to worsen and to intervene in a way that would slow or prevent progression of disease. Unfortunately, there is little known of the natural history of feline HCM and as yet no published evidence that medical therapy can alter the course of subclinical disease. The use of beta-blockers, such as atenolol, is often advocated particularly in patients that have resting LVOTO, but the efficacy of

## **Prognosis/natural history**

Survival data obtained from retrospective evaluation of teaching hospital records have been reported by two groups of investigators.<sup>29, 30</sup> Survival times for the entire study samples were similar for both studies and were close to 700 days. Median survival times of 92 days and 563 days were reported for patients with heart failure. The retrospective nature of the studies makes it difficult to interpret these differences but it seems that patients with heart failure in general, fare poorly.

## **Recommended articles (6)**