Feline hypertrophic cardiomyopathy: an update

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Section snippets

Definition

The term *hypertrophic cardiomyopathy* refers to a primary myocardial disease characterized by a hypertrophied nondilated left ventricle [1], [4]. Furthermore, HCM occurs in the absence of other cardiac diseases that might be expected to cause left ventricular hypertrophy. Finally, because HCM is classified as a primary myocardial disease, other noncardiac causes of left ventricular hypertrophy must also be excluded.

Natural history

Early reports of cats with HCM were necropsy based and reflected a dramatic disease manifesting congestive heart failure (CHF) and arterial thromboembolism (ATE) [1], [11]; however, with increasing accessibility to echocardiography, asymptomatic cats are routinely identified with HCM [3], [4], [8], [9]. Small families have been identified and clinically followed over time and confirm the heterogeneity of the disease in terms of survival and clinical manifestations [6], [9], [12]. Although it

Pathogenesis

It is reasonable to consider whether the observed heterogeneity in this disease might be explained by different etiologies. Although no disease-causing mutation has yet been identified among families of cats with HCM, there is evidence of Mendelian heritability in several pedigrees (Jo Arthur, MA, VetMB, MRCVS, personal communication, January 1997) [7], [15]. So, some percentage of feline HCM is familial; what is not known is if there are other important nongenetic etiologies of HCM. There have

Diagnosis

Antemortem diagnosis is usually made by echocardiography, with laboratory tests to exclude other systemic diseases that might cause similar cardiac changes (eg, primary hypertension, hyperthyroidism). By convention, left ventricular and interventricular septal thicknesses equal to or exceeding 6 mm are used in the diagnosis of HCM [2], [3], [4], [5]. The histologic gold standard for HCM continues to be myocyte and myofiber disarray [5].

Because HCM is believed to be predominantly a disease of

Therapy

Until recently, there was no randomized, prospective, double-blind study addressing therapy in cats with HCM. The Multicenter Feline Chronic Heart Failure Study Group conducted a study on cats with HCM that had suffered an episode of diastolic HF; however, some 20% of the cats included in this study had restrictive cardiomyopathy or feline unclassified cardiomyopathy [10]. Cats suffering concurrent thromboembolic complications were specifically excluded from the study. There was a placebo

Summary

HCM continues to be a challenging disease for veterinarians. Acute cases with ATE or CHF are difficult to manage, and we still lack the tools to advise owners well with regard to their pet's prognosis. Nevertheless, it appears that the historical view of HCM as a serious disease with a poor prognosis is now being adjusted to accommodate the apparently large numbers of asymptomatic cats with much longer survival times. Although there is evidence of a genetic cause of the disease in at least

Cited by (25)

Prospective echocardiographic and tissue Doppler screening of a large Sphynx cat population: Reference ranges, heart disease prevalence and genetic aspects

2012, Journal of Veterinary Cardiology

In the present study, HCM (including various echocardiographic LV hypertrophic patterns) was detected in 23 of the 114 cats investigated (20.2%), and was the only acquired heart disease diagnosed in the study population. This confirms that the Sphynx cat, like other feline breeds,7–16,23 is highly predisposed to HCM, which is characterized by a heterogeneous phenotypic expression. As previously reported,8,12,13 most HCM cats were young adults (mean age of 4.2 years) with a significant increase in prevalence according to age but a wide age range at diagnosis (1–10 years) and at death (4.5–11 years).

Status of therapeutic gene transfer to treat cardiovascular disease in dogs and cats

2011, Journal of Veterinary Cardiology

Recently, we analyzed myocardial samples from canine and feline patients with spontaneous heart

disease (degenerative valve disease and idiopathic dilated cardiomyopathy in dogs; hypertrophic and unclassified cardiomyopathy in cats) and observed altered expression of several proteins involved in calcium homeostasis including: s100a1, SERCA2a, and phosphorylated PLB suggesting manipulation of these pathways may be therapeutic in these animals as well (Sleeper, unpublished data). Idiopathic dilated cardiomyopathy (DCM) and chronic valve disease are the two most common acquired heart diseases in the dog, while hypertrophic cardiomyopathy is the most common form of acquired heart disease in the cat.46–52 Certain breeds are over-represented and etiologic mutations have been identified in some families of dogs and cats.53–56

Prevalence of the MYBPC3-A31P mutation in a large European feline population and association with hypertrophic cardiomyopathy in the Maine Coon breed

2010, Journal of Veterinary Cardiology

Hypertrophic cardiomyopathy has been described in several animal species, particularly in cats and dogs.10,11 Hypertrophic cardiomyopathy is the most common feline heart disease and is also a major cause of morbidity and mortality associated with a high risk of sudden death, congestive heart failure, and aortic thromboembolism in this species.1,2,12 Feline HCM is currently diagnosed by two-dimensional (2D) and M-mode echocardiography demonstrating a variety of global or regional hypertrophic patterns, predominantly involving the interventricular septum (IVS) and the left ventricular free wall (LVFW).

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