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Right ventricular involvement in feline hypertrophic cardiomyopathy

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Abstract *Objectives:* To evaluate right ventricular (RV) wall thickness and chamber dimensions in cats with hypertrophic cardiomyopathy (HCM).
Animals: One hundred fifty-one healthy control cats and 200 cats with HCM.
Methods: Retrospective, observational, clinical cohort study. Two-dimensional echocardiograms from all cats were analyzed. Right atrial diameter, RV free wall thickness, and RV chamber diameter were quantified using multiple imaging views. Conventional (mean ± 2 standard deviations) and allometrically scaled ($Y = a \times M^b$) reference values were determined in normal cats and compared to values found in cats with HCM. Linear and logistic regression, multivariate regression, and mixed model analysis were performed to identify associations between RV wall thickness and severity of left ventricular (LV) hypertrophy, clinical severity of HCM, and presence of pleural effusion.
Results: Mean RV wall thickness was increased in HCM ($p<0.001$). Considering increased RV wall thickness in at least one segment, 94 (47%) and 112 (56%) cats with HCM had RV hypertrophy using upper reference limits based on mean + 2 standard deviations or allometric scaling, respectively. There was an association between severity of LV and RV hypertrophy ($p<0.05$). Left-sided congestive heart failure (n = 58) was associated with increased RV wall thickness in all segments compared to cats with preclinical HCM ($p<0.001$). Body weight had negligible effects on RV wall thickness (R^2 0.08–0.17, $p<0.001$), whereas age and breed had no effect ($p>0.05$) in control cats.

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Conclusions: Increased RV wall thickness is common in cats with HCM and relates to severity of LV hypertrophy and clinical status.

Abbreviation

ATE	arterial thromboembolism
CHF	congestive heart failure
CV	coefficient of variation
HCM	hypertrophic cardiomyopathy
IVSd	thickness of the interventricular septum in diastole
LA4ch	left apical four-chamber view
LRL	lower reference limit
LV	left ventricular
LVDd	left ventricular internal dimension in diastole
LVH	left ventricular hypertrophy
LVPWd	thickness of the left ventricular posterior wall in diastole
Max LAD	maximum left atrial dimension
Max RAD	maximum right atrial dimension
MRI	magnetic resonance imaging
NT-proBNP	N-terminal pro brain natriuretic peptide
RPLax	right parasternal long-axis view
RPSax	right parasternal short-axis view
RVDd	right ventricular dimension in diastole
RVFWd	thickness of the right ventricular free wall in diastole
RV	right ventricular
RVH	right ventricular hypertrophy
2D	two-dimensional
URL	upper reference limit

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common heart disease in cats, leading to adverse outcomes including congestive heart failure (CHF), arterial thromboembolic disease, and cardiac death. The generally accepted definition in people describes HCM as a primary myocardial disease morphologically limited to the left ventricle [1,2] and characterized by unexplained hypertrophy in the absence of another cardiac or systemic disorder capable of producing a similar magnitude of myocardial hypertrophy [3]. Clinically, left ventricular (LV) hypertrophy (LVH) in cats is recognized by a diffuse or segmental increase of end-diastolic wall thickness $\geq$6 mm [4] with some cardiologists using 5.5 mm as a diagnostic cut-off in cats [5–7]. However, HCM is a heterogeneous disease, with a wide array of morphological, hemodynamic, neurohormonal, electrocardiographic, functional, and clinical patterns. Whether right ventricular hypertrophy (RVH) is present in feline HCM is undetermined. Right ventricular involvement in human HCM has been reported in up to two thirds of the patients [8–11]. Although unknown it has been speculated that LVH and RVH may be manifestations of the same pathophysiological process with an association between severity of left heart disease and the extent of RV wall thickening [8,11,12]. In contrast, disproportionate severe RVH in the absence of moderate and severe LVH or pulmonary hypertension secondary to left heart disease has also been documented in people with HCM [9,12,13]. Finally, it seems attractive to believe that RV involvement in the cardiomyopathic process may contribute to the development of pleural effusion as a manifestation of congestive heart failure, commonly observed in cats with HCM. To the authors' knowledge, RV morphology has not been studied comprehensively in cats. Therefore, the aim of this study was to characterize the right ventricle by two-dimensional (2D) echocardiography in large cohorts of healthy cats and cats with HCM. We hypothesized that increased RV wall thickness is common in feline HCM, is related to the severity of LVH, and if severe, is associated with clinical signs of CHF.

Animals, materials and methods

Case selection

Due to the nature of this study, no institutional animal care and use approval or client consent was sought. Medical records from May, 2003 until July, 2014 were searched for cats with the diagnosis of 'HCM', 'hypertrophic obstructive cardiomyopathy', or 'normal cardiovascular findings' that had undergone transthoracic echocardiography. Healthy cats were primarily imaged at the request of breeders, due to the presence of a soft heart murmur that required evaluation, or as part of a pre-anesthetic exam. Cats with HCM were imaged

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