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Echocardiographic characterization of a hypertrophic cardiomyopathy phenotype in an apparently healthy cat†

Arni Diana Fitri[1,*], Dwi Utari Rahmiati[1], Fitria Senja Murtiningrum[1], Leni Maylina[2], Havan Yusuf[3], Arifin Budiman Nugraha[4], Amaq Fadholly[5], Lina Noviyanti Sutardi[6], Aulia Andi Mustika[5], Vera Nora Indra Astuti[6], Zukhri Agusty Leo Syamsul Arifin[6], Amrozi[7]

[1] Division of Surgery and Radiology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[2] Division of Internal Medicine, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[3] Division of Veterinary Public Health and Epidemiology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[4] Division of Parasitology and Medical Entomology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[5] Division of Pharmacology and Toxicology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[6] Sub-Division of Veterinary Pharmacy, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[6] Administration of School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia
[7] Division of Reproduction and Obstetrics, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia

ABSTRACT: Feline hypertrophic cardiomyopathy can remain clinically silent, complicating recognition in animals with limited medical histories. This report describes echocardiographic findings in an apparently healthy male domestic shorthair stray cat, estimated to be older than five years and weighing 4.6 kg. Unsedated B-mode, M-mode, and color Doppler examinations revealed left ventricular and papillary muscle thickening. The end-diastolic interventricular septal and posterior wall thicknesses were 6.0 and 8.7 mm, respectively. The device-reported ejection fraction and fractional shortening were 93% and 59%, respectively. Mild mitral leaflet thickening was observed without detectable regurgitation. These findings support a hypertrophic cardiomyopathy phenotype, but systemic hypertension and hyperthyroidism were not excluded because blood pressure and total thyroxine were not measured.

Keywords:
feline, myocardial thickening, papillary muscles, mitral apparatus, subclinical disease

■ INTRODUCTION

Hypertrophic cardiomyopathy (HCM) can occur in cats without clinical signs. In CatScan, Payne et al. (2015) identified HCM in 14.7% of 780 apparently healthy cats after excluding hypertension and hyperthyroidism. Thus, an unremarkable appearance does not establish health. The American College of Veterinary Internal Medicine consensus (Fuentes et al. 2020) distinguishes an echocardiographic HCM phenotype from an etiological diagnosis, which is relevant in stray cats with incomplete histories. This report describes ventricular hypertrophy in an apparently healthy stray cat and examines what measurements establish about morphology, cause, and clinical risk.

■ CASE

History and physical examination. An adult male domestic shorthair cat (>5 years old, 4.6 kg) underwent echocardiographic evaluation. Physical examination revealed a body condition score of 5/9, rectal temperature of 38.5 °C, heart rate of 180 beats/min, and respiratory rate of 40 breaths/min. Hydration status was normal, with no dyspnea or fatigue observed.

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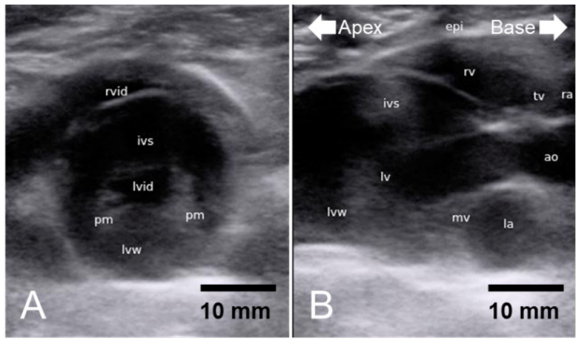


Figure 1. B-mode echocardiograms showing feline left ventricular hypertrophy (HCM phenotype). (A) Right parasternal short-axis view demonstrating severe wall and papillary muscle thickening with luminal reduction. (B) Right parasternal long-axis view demonstrating marked septal and posterior wall thickening. Note: Ao = aorta; Apx = apex; Epi = epicardium; IVS = interventricular septum; LA = left atrium; LV = left ventricle; LVID = left ventricular lumen; LVW = left ventricular wall; MV = mitral valve; PM = papillary muscle; RA = right atrium; RV = right ventricle; RVID = right ventricular lumen; TV = tricuspid valve.

Diagnostic assessment. Unsedated echocardiography in lateral recumbency was performed using the Chison Q8 system and linear transducer. Right parasternal long- and short-axis and left apical views were acquired. B-mode confirmed left ventricular and papillary muscle thickening, hyperechoic endocardial lining, and mild mitral leaflet thickening. M-mode quantified ventricular, left atrial, and aortic dimensions (Table S1&S2); color Doppler confirmed no valvular regurgitation (Table S3). Heart rate was 214 beats/min.

Diagnosis, prognosis, and management. Echocardiography confirmed left ventricular hypertrophy consistent with the HCM phenotype. Differentials include hypertensive or hyperthyroid remodeling, hypersomatotropism, aortic stenosis, pseudohypertrophy, and transient myocardial thickening. A systemic workup with serial echocardiography is required for staging, prognosis, and management.

■ RESULTS AND DISCUSSION

The primary finding was disproportionate posterior wall thickening despite stability. The ≥6 mm consensus threshold (Fuentes et al. 2020) confirms hypertrophy in normal-sized cats. Both diastolic thicknesses met this threshold, exceeding the values reported by Noviana and Kurniawan (2013). Furthermore, the 8.7-mm posterior wall measurement exceeded the ≥7 mm threshold for severe hypertrophy (Kittleson & Côté 2021). Reviewing loops and repeating measurements will confirm the findings before staging.

Marked wall thickening identifies hypertrophic cardiomyopathy (HCM) as the primary diagnosis, requiring evaluation for secondary causes. According to Kittleson and Côté (2021), hypertension, hyperthyroidism, hypersomatotropism, and aortic stenosis require exclusion. Normal hydration excludes dehydration-associated pseudohypertrophy; blood pressure, total thyroxine, and serial echocardiography assess persistence. Mitral thickening without regurgitation does not indicate myxomatous degeneration or endocardiosis (Schober & Todd 2010).

Elevated ejection fraction and fractional shortening demonstrate hyperdynamic cavity emptiness. However, preserved systole coexists with impaired relaxation in HCM; therefore, these indices do not indicate normal diastolic function (Kittleson & Côté 2021). Restraint-induced tachycardia altered measurements. Pending verification against vendor algorithms and unrounded data, dimensions remain references. A left atrial-to-aortic ratio of 1.2 alone is insufficient to confirm low risk. Standardized reassessment must precede B1/B2 staging, treatment selection, and prognostic determination.

■ CONCLUSION

Echocardiography confirmed left ventricular hypertrophy consistent with HCM in an asymptomatic stray cat. Mild mitral thickening without endocardiosis. Further evaluation will determine the etiology and clinical risk.

■ AUTHOR INFORMATION

Corresponding Author

*ADF: arnifi@apps.ipb.ac.id

Division of Surgery and Radiology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, INDONESIA.

■ ASSOCIATED CONTENT

Supporting Information

†The echocardiography measurement and parameters with 2D-Motion mode were submitted in PDF form as supporting information.

■ ETHICS AND DECLARATIONS

Conflict of interest: The authors declare no conflict of interest.
Owner consent: Informed consent was obtained from the animal owners prior to clinical procedures and data collection.
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Data availability: All data are included within the article.

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