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Doppler Echocardiographic Evaluation of Midventricular Obstruction in Cats with Hypertrophic Cardiomyopathy

H.B. MacLea, J.A. Boon, and J.M. Bright

Background: Hypertrophic cardiomyopathy (HCM) is heterogeneous in both people and cats, with variability in the distribution of hypertrophy, hemodynamic characteristics, and Doppler echocardiographic findings.

Objectives: To document the Doppler echocardiographic characteristics of midventricular obstruction in some cats with HCM.

Animals: Eight cats with hypertrophic cardiomyopathy.

Materials and Methods: Retrospective case series. The medical records of cats presenting to the cardiology service at Colorado State University between February 2009 and January 2012 were reviewed. All cats had a physical examination; Doppler systolic blood pressure measurement; and transthoracic two-dimensional (2D), M-mode, and Doppler echocardiography were performed. A more thorough evaluation of the echocardiographic images and measurements was performed. Cats included in this study had echocardiograms of adequate quality to confirm the diagnosis of midventricular obstruction by documentation of left midventricular concentric hypertrophy; a midventricular turbulent Doppler color flow pattern; and high velocity, late-peaking flow at the area of turbulence. Cats with evidence of systemic hypertension defined as a systolic Doppler blood pressure of greater than 170 mmHg were excluded.

Results: All 8 cats had left ventricular hypertrophy at the level of the papillary muscles; left, midventricular hypertrophy; and in 4/8 cats there was apical hypertrophy or basilar hypertrophy of the interventricular septum. Color flow Doppler revealed turbulent flow in 8/8 cats and spectral Doppler (continuous and pulsed wave) revealed increased flow velocities and late-peaking flow profiles at the level of the left midventricle. Two of 8 cats had a bifid midventricular flow profile in which there was a midsystolic decline in left ventricular velocities with elevated velocities extending into early diastole. The peak left ventricular outflow velocity in all 8 cats was normal.

Conclusions and Clinical Importance: A variant of HCM characterized by hypertrophy at the level of the papillary muscles with midventricular obstruction is present in some cats. Recognition of this variant of feline HCM allows identification of HCM in cats with murmurs where the more classic features of HCM are not present.

Key words: Echocardiography; Feline; HCM; Midventricle.

Hypertrophic cardiomyopathy (HCM) is the most common cardiac disorder in cats and is defined as a primary disease of the cardiac muscle characterized by concentric hypertrophy.[1–5] HCM is heterogeneous in both people and cats with variability in distribution of hypertrophy and variability in the hemodynamic characteristics among each type.[6–12] Several uncommon phenotypic variants of HCM in people include discrete apical HCM, apical HCM with extension into the mid left ventricle, and midventricular HCM with obstruction because of papillary muscle hypertrophy, producing an apical, akinetic "chamber".[7–11] Human patients with midventricular hypertrophy and obstruction have more severe clinical signs, greater frequency of ventricular arrhythmias, and typically a worse clinical outcome than patients with apical hypertrophy.[7,13] Standard M-mode echocardiography provides measurement of left ventricular chamber dimensions and wall thickness at a single, defined area near the base of the septum and, therefore, might fail to demonstrate focal midventricular hypertrophy and obstruction as the cause of a murmur.[8,10] Two-dimensional echocardiographic studies of human patients with midventricular obstruction demonstrate significant hypertrophy at the level of the papillary muscles with color flow Doppler turbulence; systolic apposition of the midventricular wall and papillary muscles; and evidence of elevated blood flow velocity persisting through late systole and often into early diastole.[7]

Abbreviations:

2D	two-dimensional
HCM	hypertrophic cardiomyopathy

In veterinary medicine, midventricular obstruction has been noted in some cats with HCM, but the Doppler echocardiographic features have not been described in detail.[6,12] The current case series characterizes the Doppler echocardiographic findings observed in 8 cats with cardiac murmurs and HCM in which murmurs resulted solely from obstruction occurring at the midventricular level.

Animals, Materials, and Methods

The echocardiographic images and measurements from cats diagnosed with midventricular obstruction at Colorado State University (CSU) between February 2009 and January 2012 were reviewed. To be included in the study, echocardiograms had to be of adequate quality to confirm the diagnosis of midventricular obstruction by documentation of left midventricular concentric

From the Department of Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO (MacLea, Boon, Bright).

Corresponding author: Dr J.M. Bright, Department of Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523; e-mail: jmbright@colostate.edu.

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hypertrophy; a midventricular turbulent Doppler color flow pattern; and high-velocity, late-peaking flow at the area of turbulence. The spectral velocity recordings were obtained using pulsed wave Doppler until an aliased signal was obtained and then confirmed with continuous wave Doppler. In addition, pulsed wave Doppler was used to document normal peak flow velocities and flow profiles at the base of the interventricular septum in the left ventricular outflow tract.

Images were recorded using a Vivid 7 Vantage™[a] and a 7S MHz Transducer[a] with harmonics. Cats were manually restrained in both right and left lateral recumbency on a padded echo table and imaged from below. The right parasternal long-axis outflow and left parasternal 5-chamber apical views were used to acquire the data, and all echocardiograms were required to have a simultaneously recorded single-lead electrocardiogram. Image analyses, measurements, and calculations were obtained from still images and cine loops stored on GE EchoPac PC Software.[a] At least 3 consecutive cardiac cycles were evaluated. Peak left ventricular outflow velocity was measured using pulsed wave Doppler with the gate placed proximal to the aortic valve. Peak velocity of flow in the left ventricular outflow tract was considered increased if greater than 1.6 m/s.[14] Late-peaking flow profiles were identified when peak velocity occurred during the last half of ejection, creating a classic dagger-shaped flow signal. Previous reports have described the left, midventricular region as the middle third of the left ventricle at the body and base of the papillary muscles.[7] Concentric hypertrophy patterns were characterized as midventricular if there was visible hypertrophy at the level of the papillary muscles. Interventricular septal thickness was considered increased if greater than 6 mm.[15] Aliasing consistent with turbulent flow in the midventricular region of the left ventricle was identified using color flow Doppler with a Nyquist limit of 0.9–1.2 m/s. Systolic left ventricular flow profiles and velocities were evaluated using pulsed wave Doppler with the sample volume placed at the level of the midventricular turbulence. If pulsed wave Doppler resulted in an aliased flow signal in the left, midventricular region, continuous wave Doppler was used to document peak velocity.

Results

Eight cats met the criteria for the case series. These cats represented 5% of the cats diagnosed with hypertrophic cardiomyopathy during the same time period at CSU. This percentage might underestimate the prevalence of cats with midventricular obstruction as the diagnosis could have been missed if the middle portion of the ventricle was not specifically evaluated with color flow and spectral Doppler. Of the 8 cats, 6 were domestic shorthair or domestic longhair, 1 was Siamese, and 1 was Persian. Cats ranged in age from 7 to 16 years (median 12.5 years) with 3 females and 5 males. All 8 cats had left-sided, parasternal, systolic cardiac murmurs ranging in intensity from grade II-IV/VI. The murmurs did not vary in intensity in individual cats. Heart rate ranged from 174 to 246 bpm (median 189 bpm). Systolic Doppler arterial blood pressure ranged from 93 to 180 mmHg (median 148.5 mmHg). In addition to cardiac disease, several cats had concurrent illness, including inflammatory bowel disease, eosinophilic granuloma complex, neurologic disease, neoplasia, chronic renal disease, hyperthyroidism, and diabetes mellitus. Treatment of cats in the study was variable and included atenolol,[b] diltiazem,[c] and heart failure management consisting of

Table 1. M-mode and 2D echocardiographic measurements of left ventricular chamber dimensions, wall thickness, and left atrial size and Doppler measurements of left midventricular and left ventricular outflow velocities in study cats.

Echo Parameter	Mid LV Obs Velocity (m/s)	Mid LV PG (mmHg)	LV Outflow Velocity (m/s)	LV Outflow PG (mmHg)	IVSd (mm)	LVPWd (mm)	AO(2D) (mm)	LA (2D) (mm)	LA/AO (2D)	AO (M-Mode) (mm)	LA (M-Mode) (mm)	LA/AO (M-Mode)	LVIDd (mm)
Median	2.22	19.70	1.43	8.15	6.30	5.63	8.29	13.80	1.68	8.73	14.6	1.52	11.55
(Range)	(1.90–3.50)	(8.00–49.0)	(1.10–1.60)	(4.80–10.2)	(4.43–8.11)	(3.78–6.96)	(7.50–9.80)	(10.13–17.90)	(1.21–2.32)	(7.50–11.4)	(11.8–18.1)	(1.27–2.22)	(9.30–16.02)

LV, left ventricular; PG, pressure gradient; Obs, obstruction; IVSd, interventricular septum diastole; LVPWd, left ventricular posterior wall diastole; AO, aorta; LA, left atrium; LVIDd, left ventricular interior diameter diastole.

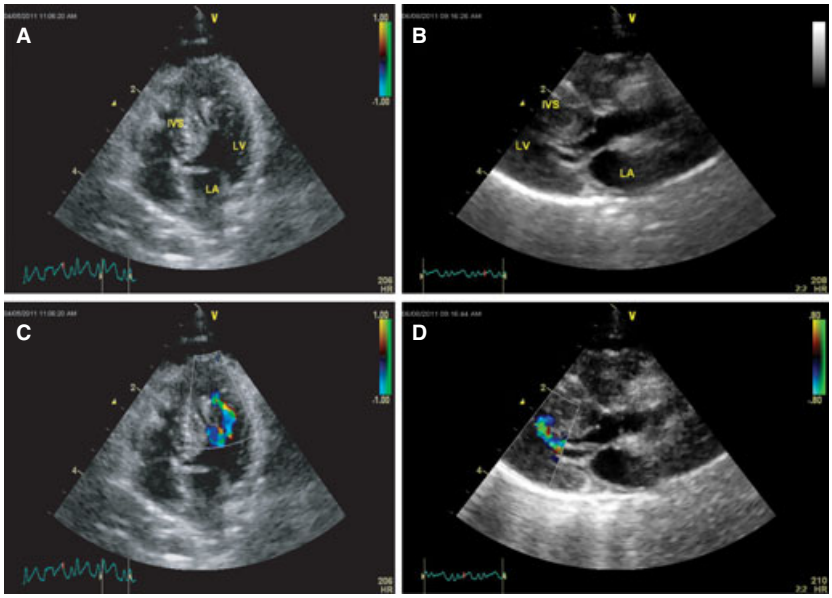

Fig 1. Two-dimensional echocardiographic images from a cat with left ventricular hypertrophy and obstruction. **(A)** Left apical 4-chamber view obtained during systole showing the interventricular septum, left atrium, and left ventricle. **(B)** Right long-axis outflow view during systole showing the interventricular septum, left atrium, and left ventricle. Note that hypertrophy of the LV wall extends from midventricle to apex and along the entire interventricular septum. **(C)** A similar image as in (A) with color flow Doppler demonstrating apical and midventricular turbulent flow. **(D)** A similar image as in (B) with color flow Doppler demonstrating turbulent flow at the level of the papillary muscles.

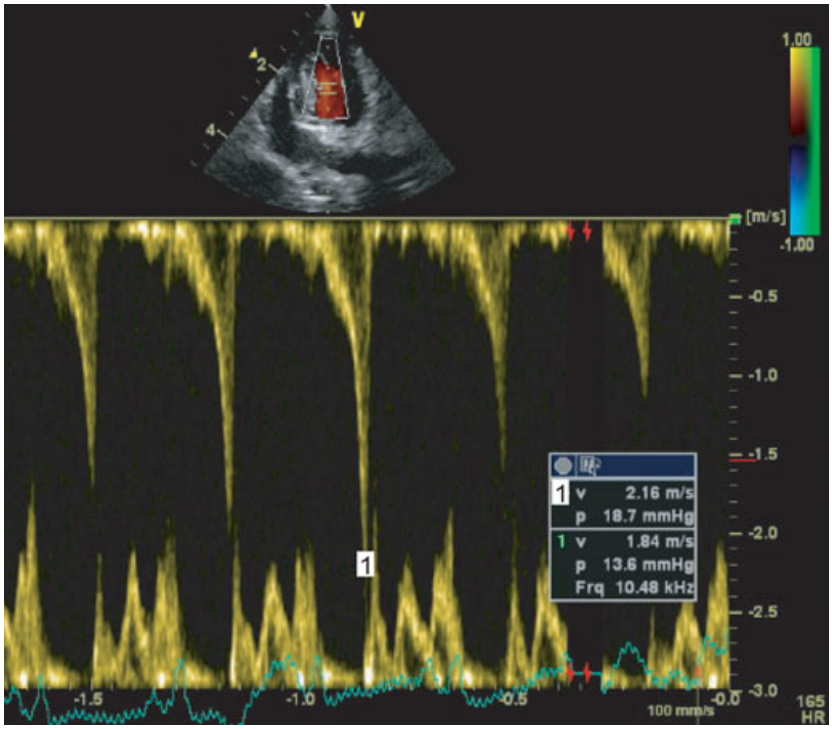

Fig 2. Left apical 4-chamber image obtained from a cat with left, midventricular obstruction. Pulsed wave Doppler sampled from the left, midventricular chamber at the level of the papillary muscles demonstrates a late-peaking flow profile with elevated peak flow velocity (labeled #1 at 2.16 m/s; PG 18.7 mmHg).

an ACE-inhibitor (benazepril[d] or enalapril[e]), antithrombotics (clopidogrel[f]), and furosemide.[g] Two cats did not receive any cardiac medications, in 1 cat because of minimal cardiac disease and the other because of concurrent neurologic disease, which resulted in euthanasia shortly after cardiac evaluation.

On 2D echocardiographic examination, distribution of left ventricular hypertrophy was variable among cats and was visible at the basilar portion of the interventricular septum, in the midventricle at the level of the papillary muscles, or extending from the left midventricle apically (Table 1, Fig 1A and B). None of the cats had an apical aneurysm. All 8 cats had hypertrophy at the level of the papillary muscles with a turbulent midventricular color Doppler flow pattern and spectral Doppler measurements confirming abnormally high velocity flow in this area of the left ventricle (Fig 1C and D). Measured midventricular velocities ranged from 1.90 to 3.50 m/s (mean 2.50 m/s) representing estimated gradients from 13.8 to 49.0 mmHg (mean 24.1 mmHg). All cats had normal left ventricular outflow velocities (peak <1.6 m/s)[14] including 4/8 cats with increased thickness at the base of the septum, and in all cats midventricular velocities exceeded left ventricular outflow velocities. Six cats had late-peaking (dagger-shaped) systolic flow at the level of the midventricle (Fig 2). The remaining 2 cats demonstrated a bifid midventricular flow profile in which there was a midsystolic decline in left ventricular velocities followed by elevated velocities extending into early diastole (Fig 3). There was no correlation between intensity of the cardiac murmurs and left midventricular flow velocities.

Discussion

Variants of HCM have been described in people and in cats. Previously reported variants in cats include symmetric LV hypertrophy, asymmetric LV hypertrophy with either increased thickness of the LV free wall or septum, and midventricular hypertrophy.[6,12] This case series illustrates that despite the presence of basilar hypertrophy in 4/8 cats, all cats had normal left ventricular outflow velocities with no evidence of systolic anterior motion of the mitral valve or mitral valve regurgitation. Furthermore, 8/8 cats had midventricular left ventricular hypertrophy, midventricular obstruction, and systolic murmurs attributed solely to midventricular turbulence.

No cat appeared to have a distal left ventricular aneurysm or apical, akinetic "chamber" as described in humans; although the true left ventricular apex might not have been visible during every echocardiographic examination. In human patients visualization of a concealed or small apical, akinetic "chamber" often requires intravenous contrast injection, which was not performed in the cats of this series.[13]

Two of 8 cats had a bifid midventricular flow profile indicating a midsystolic decline in left ventricular flow velocities with elevated velocities in early systole and in late systole continuing into early diastole. This

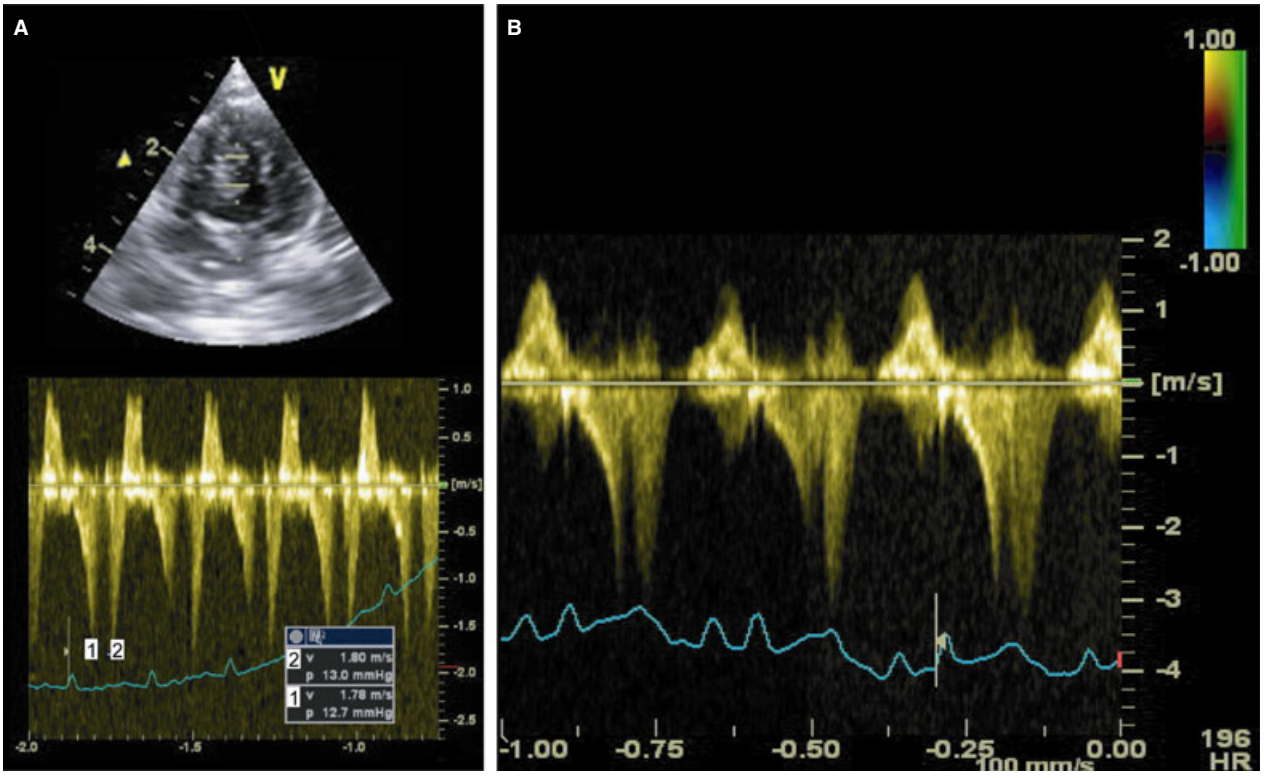

Fig 3. Spectral Doppler images obtained from 2 cats with midventricular hypertrophy and obstruction. **(A)** Pulsed wave Doppler sampled from the left, midventricular chamber at the level of the papillary muscles demonstrating an initial increase in velocity (labeled #1 at 1.78 m/s; PG 12.7 mmHg) followed by a marked decrease in midsystolic velocity and a 2nd peak of even higher velocity extending into the isovolumic relaxation (labeled #2 at 1.80 m/s; PG 13.0 mmHg). **(B)** Continuous wave Doppler obtained from the left apical position showing increased early systolic flow velocity followed by a midsystolic decline in velocity and abnormally high velocities extending into early diastole.

Doppler finding in human patients is attributed to the presence of an apical, akinetic "chamber" that arises as a result of the midventricular hypertrophy with resultant intraventricular afterload mismatch during peak systole.[7] The apical segments cannot maintain flow against the midsystolic rise in afterload that develops early in systole; however, flow accelerates late in systole as the hypertrophied myocardium relaxes. It is possible, therefore, that an apical, akinetic "chamber" was present but could not be imaged in the absence of administration of a contrast agent in the 2 patients with bifid flow profiles.

Recognition of the Doppler echocardiographic findings of midventricular hypertrophy and midventricular obstruction of the left ventricle in cats could have therapeutic and prognostic implications. The cats in this case series had normal left ventricular outflow velocities and no evidence of systolic anterior motion, secondary mitral valve regurgitation, late-peaking left ventricular outflow velocity, or dynamic right ventricular outflow obstruction. Absence of these more typical echocardiographic features of feline HCM may potentially result in misdiagnosis of cats with audible cardiac murmurs. It could also be therapeutically important to recognize left, midventricular obstruction, as dynamic obstruction increases wall stress and myocardial oxygen demand, and may be reduced by the administration of beta-adrenergic antagonists or calcium channel blockers.[16] Long-term follow-up of cats with left, midventricular obstruction is needed to determine whether clinical signs, when present, become more severe than in cats with the classic form of subaortic obstructive HCM as described in humans.

Footnotes

[a] GE Medical Systems, Milwaukee, WI
[b] Atenolol; Ranbaxy Pharmaceuticals, Jacksonville, FL
[c] Diltiazem CD; Actavis-Elizabeth, LLC., Morristown, NJ
[d] Benazepril Hydrochloride; Ranbaxy Pharmaceuticals
[e] Enalapril Maleate, Wockhardt USA/LLC., Parsippany, NJ
[f] Plavix, Clopidogrel Bisulfate, Bristol-Myers Squibb/Sanofi, Bridgewater, NJ
[g] Salix, Furosemide; Intervet Inc, Summit, NJ

Acknowledgment

Conflict of Interest Declaration: Authors disclose no conflict of interest.

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