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Early detection of myocardial dysfunction using two-dimensional speckle tracking echocardiography in a young cat with hypertrophic cardiomyopathy

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Abstract

Case summary A 5-month-old intact female Scottish Fold cat was presented for cardiac evaluation. Careful auscultation detected a slight systolic murmur (Levine I/VI). The findings of electrocardiography, thoracic radiography, non-invasive blood pressure measurements and conventional echocardiographic studies were unremarkable. However, two-dimensional speckle tracking echocardiography revealed abnormalities in myocardial deformations, including decreased early-to-late diastolic strain rate ratios in longitudinal, radial and circumferential directions, and deteriorated segmental systolic longitudinal strain. At the follow-up examinations, the cat exhibited echocardiographic left ventricular hypertrophy and was diagnosed with hypertrophic cardiomyopathy using conventional echocardiography.

Relevance and novel information This is the first report on the use of two-dimensional speckle tracking echocardiography for the early detection of myocardial dysfunction in a cat with hypertrophic cardiomyopathy; the myocardial dysfunction was detected before the development of hypertrophy. The findings from this case suggest that two-dimensional speckle tracking echocardiography can be useful for myocardial assessment when conventional echocardiographic and Doppler findings are ambiguous.

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Introduction

Hypertrophic cardiomyopathy (HCM), a primary disorder of the myocardium, is the most common cardiac disease in cats.[1,2] However, obtaining a diagnosis of HCM in cats is occasionally difficult, particularly in the absence of left ventricular (LV) hypertrophy.[2] Recently, two-dimensional (2D) speckle tracking echocardiography (2D-STE) was used for the diagnosis and assessment of HCM in humans and cats.[3–8] Previous feline studies demonstrated that 2D-STE detects myocardial dysfunction in cats with HCM that exhibit echocardiographic evidence of LV hypertrophy.[5–8] This technique has enabled the assessment of myocardial variables such as strain and strain rate, which provide better quantification of regional and global myocardial deformations, and may have higher sensitivity than conventional echocardiographic parameters for the detection of subtle

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Table 1 Conventional two-dimensional and Doppler echocardiography data at first visit (day 0) and follow-up (days 90 and 150) examinations

	Day 0	Day 90	Day 150
Left atrial to aortic root ratio	1.5	1.5	1.6
End-diastolic interventricular septum thickness (mm)	4.5	4.6	5.0
End-diastolic left ventricular free-wall thickness (mm)	2.7	3.9	4.3
End-diastolic left ventricular internal dimension (mm)	12.2	14	14.1
Relative wall thickness	0.59	0.60	0.66
End-diastolic subaortic interventricular septum thickness (mm)	4.9	5.9	7.2
Left ventricular outflow tract velocity (m/s)	0.8	1.8	3.9
Transmitral E and A ratio	1.1	Fusion	1.4
Doppler signal of the left ventricular outflow tract	Laminar	Intermittently turbulent	Turbulent
Presence of systolic anterior motion	Absent	Intermittent	Present
Presence of mitral regurgitation	Absent	Intermittent	Present
Presence of papillary muscle hypertrophy	None	Equivocal	Obvious

myocardial dysfunction.[9–11] Human studies demonstrated that 2D-STE detects myocardial dysfunction in preclinical HCM mutation carriers with normal LV wall thickness.[12,13] Here we describe the early detection of myocardial dysfunction before the occurrence of LV hypertrophy using 2D-STE in a young cat with HCM.

Case description

A 5-month-old intact female Scottish Fold cat weighing 2.1 kg was presented at Nippon Veterinary and Life Science University for consultation of cardiac murmur. The cat had no history of clinical signs. Careful auscultation detected a slight systolic murmur (Levine I/VI). Electrocardiography, thoracic radiography and non-invasive blood pressure measurements (systolic blood pressure 112 mmHg) were normal.

At the first visit (day 0), conventional 2D and Doppler examinations were performed using a Vivid 7 echocardiographic system (GE Healthcare). The cat was not sedated, but was manually restrained in the right and left lateral recumbent positions. LV wall thickness was measured by 2D methods. The end-diastolic interventricular septum thickness was 4.5 mm, end-diastolic LV free-wall thickness was 2.7 mm, end-diastolic LV internal dimension was 12.2 mm, end-systolic LV internal dimension was 4.9 mm and fractional shortening was 50.4% on a short-axis view. All wall thicknesses, including the subaortic interventricular septum wall thickness on a long-axis view of 4.9 mm, were <6 mm, indicating the absence of LV hypertrophy. The left atrial to aortic root ratio derived from the 2D short-axis view was 1.6. Transmitral E- and A-wave velocities were 0.68 and 0.61 m/s, respectively. The deceleration time for the E-wave was 101 ms. The peak velocity of the LV outflow tract at rest was 0.8 m/s. This value slightly increased on excitation (1.2 m/s), although it was not found to be turbulent by pulse-wave Doppler methods. Obvious systolic anterior motion of the mitral valve or mitral regurgitation was not detected. Conventional 2D and Doppler echocardiography did not reveal any sign of HCM (Table 1).

High-quality images for 2D-STE analysis were carefully obtained by the same investigator. Images were analysed using an offline EchoPAC workstation (GE Healthcare), as described previously.[7,8,11,14,15] We measured the peak global and segmental systolic (S) strain and strain rate and the peak early diastolic (E) and late diastolic (A) strain rate in the longitudinal, circumferential and radial directions. The mean values of measurements for three consecutive cardiac cycles obtained from high-quality images were used in all analyses. Observer variability of 2D-STE analysis in our laboratory was previously described.[7,8,11,14,15] Healthy cat ranges were established from a population of 14 young healthy cats (median age 10.0 months, median body weight 3.5 kg) as in this report. These cats are part of our previously published controls.[7,8]

Diastolic 2D-STE deformations on day 0 were dramatically different from healthy cats data (Table 2). The early diastolic strain rate (peak E) in the radial and circumferential directions was decreased. The early-to-late diastolic strain rate ratio (E:A) in the longitudinal, radial and circumferential directions was decreased. Although the global peak systolic 2D-STE variables on day 0 were within the healthy cats ranges (Table 3), post-systolic shortening during the diastolic phase (Figure 1) and lower and non-synchronous segmental strains (basal septum) corresponding to the gradually hypertrophied segments on follow-up examinations (subaortic interventricular septum) were observed (Table 4).

Conventional 2D and Doppler examinations, and 2D-STE analysis were performed on days 90 and 150 using the same echocardiographic system by the same investigator. The LV wall thickness of the cat had

Table 2 Peak early (E) and late diastolic (A) strain rates assessed by two-dimensional speckle-tracking echocardiography at first visit (day 0) and follow-up (days 90 and 150) examinations

	Day 0	Day 90	Day 150	Healthy cats
Longitudinal strain rate				
Peak E (1/s)	4.3	3.2*	3.5	5.4 (3.4–7.1)
Peak A (1/s)	3.9	1.9	2.7	3.0 (1.0–5.1)
E:A	1.1*	1.7	1.3	1.7 (1.2–4.0)
Radial strain rate				
Peak E (1/s)	–4.7*	–3.9*	–2.0*	–10.1 (–6.2 to –13.1)
Peak A (1/s)	–5.4	–5.0	–3.3	–4.8 (–1.0 to –11.1)
E:A	0.87*	0.79*	0.66*	2.4 (1.8–6.0)
Circumferential strain rate				
Peak E (1/s)	2.0*	2.2*	2.0*	4.7 (2.8–7.2)
Peak A (1/s)	1.8	1.9	2.0	1.9 (0.8–3.5)
E:A	1.1*	1.2*	1.0*	2.3 (1.4–5.0)

Healthy cat ranges (median and ranges) were established from a population of young healthy cats
*Values outside minimum–maximum ranges from the data of healthy cats

Table 3 Peak systolic global strain and strain rate assessed by two-dimensional speckle-tracking echocardiography at first visit (day 0) and follow-up (days 90 and 150) examinations

	Day 0	Day 90	Day 150	Healthy cats
Longitudinal strain (%)	–22.8	–21.2	–17.7*	–21.8 (–18.6 to –32.1)
Longitudinal strain rate (1/s)	–3.2	–4.2	–3.2	–3.5 (–2.0 to –4.9)
Radial strain (%)	69.9	50.4	29.3*	68.8 (33.2–98.4)
Radial strain rate (1/s)	6.2	6.2	3.7	7.1 (2.7–10.3)
Circumferential strain (%)	–23.0	–17.4*	–15.7*	–24.0 (–19.3 to –26.0)
Circumferential strain rate (1/s)	–2.8	–2.5	–2.2	–3.2 (–2.2 to –3.7)

Healthy cat ranges (median and ranges) were established from a population of young healthy cats
*Values outside minimum–maximum ranges from the data of healthy cats

gradually increased (Table 1) and the papillary muscles had subjectively hypertrophied. On day 150, the end-diastolic subaortic interventricular septum thickness was 7.2 mm. Obvious systolic anterior motion of the mitral valve with mitral regurgitation and LV outflow tract obstruction (peak velocity of LV outflow tract was 3.9 m/s, with a turbulent Doppler signal) were also observed. We identified LV hypertrophy and made a clinical diagnosis of HCM. However, fractional shortening assessed by 2D methods was 37.1%. The left atrial to aortic root ratio derived from the 2D short-axis view was 1.6. Transmitral E and A wave velocities were 0.92 and 0.67 m/s, respectively. The deceleration time of the E wave was 113 ms.

On day 150, 2D-STE revealed a decrease in the systolic global strains in the longitudinal, radial and circumferential directions (Table 3). Diastolic deformations were also observed (Table 2). Furthermore, the basal

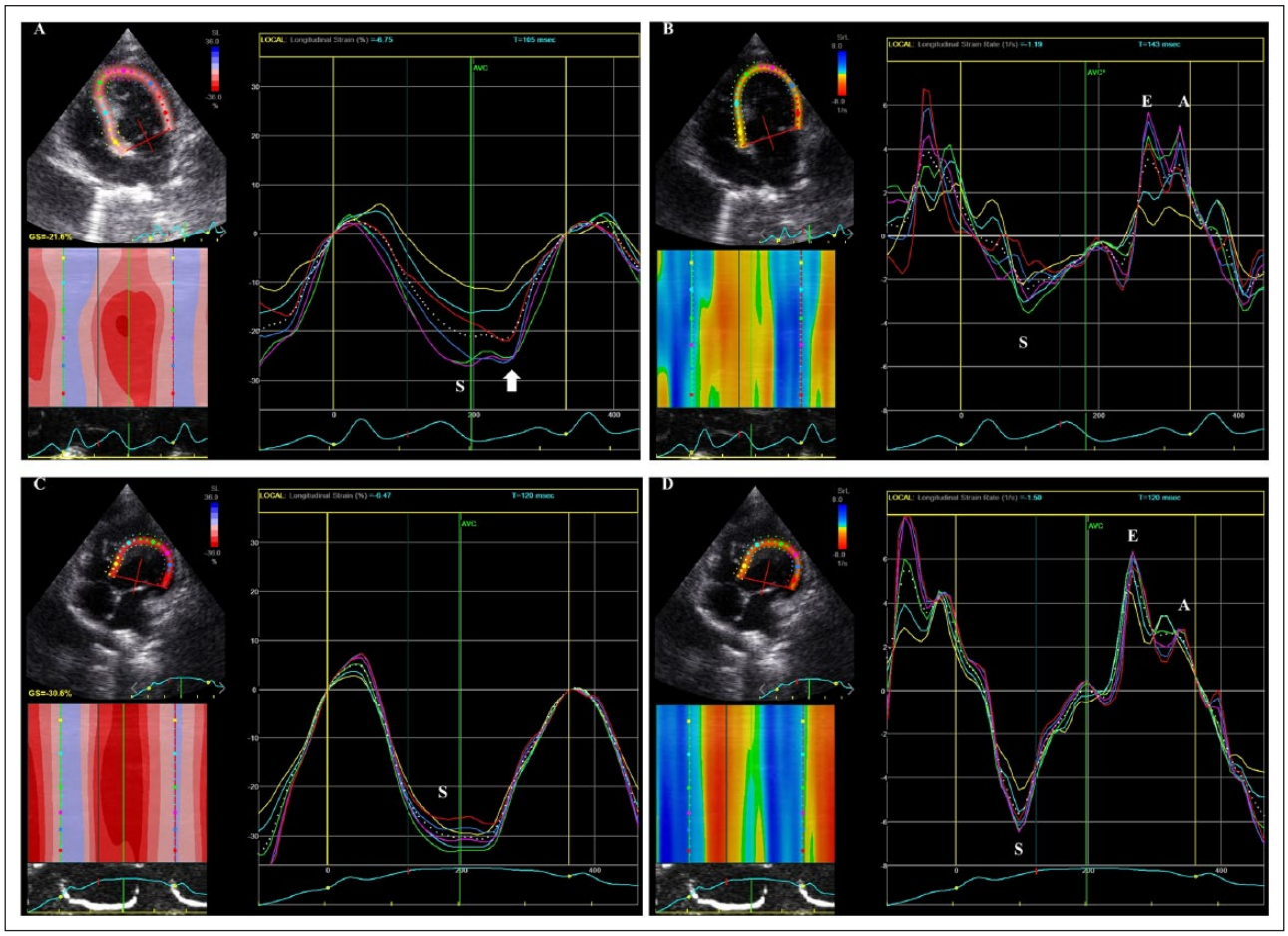

Figure 1 (a) Time-longitudinal global (dotted line) and segmental (coloured lines) longitudinal strain curve obtained from two-dimensional speckle tracking echocardiography (2D-STE; left apical four-chamber view) on day 0 in a young cat with hypertrophic cardiomyopathy (HCM). Six segmental curves are designated as the basal septum (yellow), middle septum (light blue), apical septum (green), apical lateral (purple), middle lateral (dark blue) and basal lateral (red) for speckle tracking analysis. Post-systolic shortening during the diastolic phase (arrow) can be observed. Note the lower and non-synchronous segmental strains (yellow and light-blue lines) corresponding to gradually hypertrophied segments on follow-up examinations (interventricular septum walls). All wall thicknesses on day 0, including the subaortic interventricular septum wall thickness on a long-axis view, were <6 mm, indicating the absence of left ventricular hypertrophy. (b) Time-longitudinal global (dotted line) and segmental (coloured lines, same as designated as above) longitudinal strain rate curve obtained from 2D-STE (left apical four-chamber view) on day 0 in a young cat with HCM. Note the small peak of systolic (S) and early diastolic (E) strain rate values. Global longitudinal strain rate E:A was 1.1. Conventional 2D and Doppler echocardiography did not reveal any sign of HCM on day 0. (c) Time-longitudinal global (dotted line) and segmental (coloured lines, same designated as above) longitudinal strain curve obtained from 2D-STE (left apical four-chamber view) for a healthy cat. Note the synchronous strains. (d) Time-longitudinal global (dotted line) and segmental (coloured lines, same designated as above) longitudinal strain rate curve obtained from 2D-STE (left apical four-chamber view) for a healthy cat. Note the strain rate E:A ratio of 1.9

septum, middle lateral and basal lateral segmental strains in the longitudinal direction exhibited a decrease (Table 4).

Discussion

We reported the early detection of myocardial dysfunction before the occurrence of LV hypertrophy using 2D-STE in a young cat with HCM. Conventional 2D and Doppler echocardiography methods are used for the clinical diagnosis of feline HCM. Usually, HCM is suspected in cats with an end-diastolic LV wall thickness of ⩾6 mm. However, such small wall measurements are sometimes wrongly estimated and lead to misdiagnosis, particularly in the early stages of the disease. Moreover, conventional 2D and Doppler echocardiography methods cannot detect HCM in cats with a normal LV wall thickness (absence of morphological hypertrophy, presence of causative mutations and functional abnormalities).

Tissue Doppler imaging has been used for the detection of myocardial dysfunction in a cat with subclinical

Table 4 Peak systolic segmental longitudinal strains assessed by two-dimensional speckle-tracking echocardiography at first visit (day 0) and follow-up (days 90 and 150) examinations

	Day 0	Day 90	Day 150	Healthy cats
Basal septum (%)	–13.4*	–11.9*	–6.3*	–21.5 (–15.2 to –26.0)
Middle septum (%)	–19.3	–21.2	–19.6	–26.0 (–19.3 to –35.2)
Apical septum (%)	–28.4	–34.3	–33.6	–30.4 (–22.6 to –44.5)
Apical lateral (%)	–28.1	–28.3	–29.4	–30.9 (–25.6 to – 40.0)
Middle lateral (%)	–26.9	–15.3*	–14.2*	–24.4 (–18.1 to –33.0)
Basal lateral (%)	–23.9	–13.1	–8.0*	–19.3 (–11.3 to –26.6)

Healthy cat ranges (median and ranges) were established from a population of young healthy cats
*Values outside minimum–maximum ranges from the data of healthy cats

(before hypertrophic phase) HCM and cats carrying HCM mutations.[16,17] However, measurements obtained using tissue Doppler imaging are affected by translation of the heart itself, tethering of surrounding myocardial motions, and Doppler angle dependency.[18,19] 2D-STE, a newly developed echocardiographic tool, tracks grey-scale B-mode images of unique speckle patterns in the myocardium; therefore, it is relatively independent of cardiac translation, tethering and angle parameters.[20] This technique has enabled the assessment of myocardial variables such as strain and strain rate, which provide better quantification of regional and global myocardial deformations and may have higher sensitivity than conventional echocardiographic parameters for the detection of subtle myocardial dysfunction.[9–11] Because the diagnosis of HCM in cats is sometimes difficult, assessment of the myocardium using 2D-STE may be useful to distinguish cats with HCM from healthy cats.

In the present case, conventional 2D and Doppler echocardiography examinations performed on day 0 did not reveal LV hypertrophy or myocardial abnormalities. However, 2D-STE identified marked myocardial dysfunction, including decreased E strain rates and E:As. These findings indicate a decrease in the rate of LV relaxation, increased myocardial stiffness and enhanced late diastolic filling, and suggest occult myocardial dysfunction in cats with HCM, as reported in previous studies.[3,4,21,22] Human studies demonstrated that 2D-STE detects myocardial dysfunction in preclinical HCM mutation carriers with normal LV wall thickness.[23,24] Histopathological changes and alterations in the myocardial fibre orientation may be related to these functional abnormalities.[25] The findings from our case suggest that myocardial abnormalities assessed by 2D-STE may help in the detection of HCM even before the occurrence of LV hypertrophy.

In the present case, conventional 2D and Doppler echocardiography performed on day 150 revealed the findings of HCM, although conventional systolic (LV internal dimension and fractional shortening) and diastolic (transmitral Doppler) assessments remained unchanged. However, global deformations in the longitudinal, radial and circumferential directions showed a decrease, as did diastolic deformations. These changes may have been altered by myocardial compensatory mechanisms and myocardial pathological changes, as observed in previous studies on cats with HCM and echocardiographic evidence of LV hypertrophy.[7,8] 2D-STE analysis allows for the non-invasive detection of abnormal systolic and diastolic deformations in cats with HCM that exhibit apparently normal systolic and diastolic findings in conventional echocardiography.

One of the segmental systolic deformations in the longitudinal direction (basal septum), which corresponded to the gradually hypertrophied wall on follow-up examinations (subaortic interventricular septum), showed a deterioration on day 0 (Table 4). This deformation may reflect local functional abnormalities in the myocardium before the occurrence of LV hypertrophy. In addition, some segmental longitudinal deformations (basal septum, middle lateral and basal lateral) were decreased on day 150, although that wall thickness had not reached 6 mm during follow-up examinations. A previous feline study demonstrated that tissue Doppler-derived longitudinal strain decreased with an increase in LV concentric hypertrophy.[26] These relationships may be explained by an association of the degree of myocardial changes and ischaemia with the severity of wall thickening.[27] Because HCM is caused by mutations in genes that encode for the myofilament sarcomeric proteins,[26]

functional abnormalities prior to LV hypertrophy may progress to morphological abnormalities. 2D-STE-derived myocardial deformations may reflect subtle functional abnormalities that are independent of morphological changes in the myocardium.[28] The relationships among myocardial deformations, myocardial function, and histopathological characteristics of the myocardial wall in cats with HCM should be investigated in future studies.

Conclusions

Even though the diagnosis of HCM in the present case was based on morphological rather than pathological criteria, we demonstrated that 2D-STE-derived myocardial deformations were altered before the occurrence of echocardiographic hypertrophy. Furthermore, 2D-STE analysis allows for the non-invasive detection of abnormal systolic and diastolic deformations in cats with HCM that exhibit apparently normal systolic and diastolic findings in conventional echocardiography. These findings suggest that 2D-STE may be a sensitive tool for the early detection of HCM in cats.

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