Tachycardia-induced cardiomyopathy in a cat

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Summary

A 10-year-old male castrated Domestic Shorthair cat was evaluated for an asymptomatic tachyarrhythmia noted two weeks prior. Electrocardiography revealed a normal sinus rhythm with atrial premature complexes and paroxysms of supraventricular tachycardia with a heart rate between 300 and 400 min-1. Echocardiography was unremarkable, and concentrations of circulating cardiac troponin I, T4, and blood taurine were within reference ranges. The cat was treated with sotalol (2.1 mg/kg q12h, PO) but the arrhythmia was insufficiently controlled as determined during several re-examinations within a two-year time period. Twenty four months after initial presentation atrial fibrillation with fast ventricular response rate (200 to 300 min⁻¹) was diagnosed, along with severe eccentric chamber remodeling and systolic dysfunction. The cat developed congestive heart failure and cardiogenic shock and was euthanized nearly 27 months after the first exam. Gross and histopathologic findings ruled out commonly seen types of primary myocardial disease in cats. The persistent nature of the tachyarrhythmia, the progressive structural and functional cardiac changes, and comparative gross and histopathologic post-mortem findings are consistent with the diagnosis of tachycardia-induced cardiomyopathy.

Keywords: feline, arrhythmia, heart, electrocardiogram

Tachykardie-induzierte Kardiomyopathie bei einer Katze

Eine 10-jährige männlich-kastrierte Kurzhaarkatze wurde wegen einer asymptomatischen Tachykardie vorgestellt, die erstmalig vor zwei Wochen diagnostiziert worden war. Bei der elektrokardiographischen Untersuchung wurde ein normaler Sinusrhythmus mit atrialen Extraystolen und Perioden von paroxysmaler supraventrikulärer Tachykardie mit einer Herzfrequenz von 300 bis 400 min⁻¹ festgestellt. Die echokardiographische Untersuchung war unauffällig und die Blutkonzentrationen von kardialem Troponin I, Gesamtthyroxin sowie Taurin lagen in den jeweiligen Normbereichen. Die Katze wurde mit Sotalol (2.1 mg/kg zweimal täglich, per os) behandelt, was allerdings nicht zur gewünschten Kontrolle der Arrhythmie führte, wie bei wiederholten Untersuchungen innerhalb einer zweijährigen Zeitperiode festgestellt wurde. 24 Monate nach der Erstuntersuchung entwickelte die Katze Vorhofflimmern mit schneller Kammerfrequenz (200 bis 300 min-1). Ausserdem lag eine schwere Dilatation der Herzkammern mit systolischer Dysfunktion vor. Die Katze entwickelte Herzinsuffizienz und kardiogenen Schock und wurde auf Wunsch des Besitzers 27 Monate nach Erstvorstellung euthanasiert. Die pathologisch-anatomische und histologische Untersuchung des Herzens war unauffällig, wobei die klassischen primären Kardiomyopathien der Katze mit Sicherheit ausgeschlossen werden konnten. Die lange Dauer der Tachyarrhythmie, die progressiven strukturellen und funktionellen Veränderungen des Herzens sowie die ähnlichen pathologischen und histologischen Befunde lassen auf das Vorliegen einer Tachykardie-induzierten Kardiomyopathie schliessen.

Schlüsselwörter: Katze, Arrhythmie, Herz, Elektrokardiogramm

Case history and clinical evaluation

A 10-year old, 5.7 kg, male castrated Domestic Shorthair cat was presented to the cardiology service at The Ohio State University Veterinary Medical Center for evaluation of an asymptomatic arrhythmia. The arrhythmia was detected 2 weeks prior by the referring veterinarian during a routine clinical evaluation and was described as intermittent prolonged periods of tachycardia with maximum heart rates between 300 and 400 min⁻¹. Physical examination revealed tachypnea at 76 breaths per minute with normal respiratory effort and lung sounds and an irregular heart rhythm with an average heart rate of 180 min⁻¹. Systolic blood pressure was 111 mmHg (reference range, 100-160; Ultrasonic Doppler flow detector, Model 811-B, Parks Medical Electronics Inc, Aloha/ OR, USA). Blood biochemical analysis revealed elevated creatine kinase (957 U/L; reference range, 70 to 550), hyperproteinemia (83 g/L; reference range, 56 to 76), hyperalbuminemia (36 g/L; reference range, 25 to 35), hyperglobulinemia (47 g/L; reference range, 31 to 41), a normal total plasma T4 concentration (21.9 nmol/L; reference range, 12.8 to 38.6), and a normal serum cardiac troponin I concentration (0.11 ng/mL; reference range, 0 to 0.11). On continuous ECG normal sinus rhythm with a heart rate of 142 min-1 with frequent atrial premature complexes, infrequent ventricular premature complexes (VPCs) of both right and left bundle branch block morphology, and paroxysms of narrow complex tachycardia at rates up to 330 min⁻¹ were found (Fig. 1 A–D) that were suspected to be supraventricular in origin, however high-ventricular ectopy could not be ruled out. Echocardiography (Vivid 7 Vantage, GE Medical Systems, Milwaukee/WI, USA) revealed mildly increased left atrial (LA) size (anteroposterior dimension 17.2 mm; reference range, 12 to 16), normal myocardial diastolic wall thickness (3.6 mm for the interventricular septum and 4.3 mm for the left ventricular (LV) posterior wall; reference range, 3 to 6 mm), normal LV and right-sided chamber dimensions and papillary muscle anatomy, normal LV systolic function as assessed by shortening fraction (48%; reference range, 35 to 55), and mild LV diastolic dysfunction based on a reduced early (Peak E) to late (Peak A) filling velocity ratio (E:A 0.64; reference range, 1.0 to 2.0; Santilli and Bussadori, 1998). The ECG during the echocardiographic study also revealed frequent runs of narrow-complex tachycardia.



Figure 1: Composite of ECG abnormalities observed during the study period. Panel A) Normal sinus rhythm. Atrial (*) and right ventricular (**) premature complexes. Panel B) Normal sinus rhythm and left ventricular premature complex (*). Panel C) Normal sinus rhythm with paroxysm of supraventricular tachycardia (multiple asterisks). Panel D) Atrial fibrillation with fast ventricular response rate (maximun 300 min⁻¹). Paper speed 25 mm/s (panels A and D) and 50 mm/s (panels B and C). Sensitivity 10mm = 1 mV (panel A) and 20 mm = 1 mV (panels B, C, and D).

Tentative diagnosis

No definitive cardiac diagnosis was determined, but idiopathic tachyarrhythmia, reciprocating atrioventricular tachycardia, an arrhythmia associated with chronic myocarditis, arrhythmogenic right ventricular cardiomyopathy (ARVC), or other unidentified myocardial disease were considered. A "working" diagnosis of supraventricular tachyarrhythmia of unknown etiology was made. The cat's ECG was observed continuously for at least 6 hours during diagnostic work-up period with repeated episodes of the arrhythmias as described prior. An ambulatory (Holter) 24h-ECG to further quantify the arrhythmia was recommended but declined by the cat owner.

Therapy

Sotalol (2.1 mg/kg q12h, PO; Sotalol generic, Teva Pharmaceuticals, Sellersville, PA, USA) was instituted and well tolerated. A regular cardiac rhythm with a heart rate of 164 min⁻¹ and infrequent premature beats was identified four hours post sotalol on day 8 after institution of medical treatment. The ECG was similar to previous recordings and revealed a normal sinus rhythm at a rate of 150 min⁻¹ with occasional atrial and ventricular premature complexes. Sotalol at the same dose was continued.

Time course and re-evaluation

Three months later the cat was doing well. Cardiac auscultation revealed an irregular rhythm with a heart rate of 128 to 136 min⁻¹. A normal sinus rhythm with a heart rate of 140 min⁻¹, occasional APCs and VPCs, and frequent short runs of narrow complex tachycardia at a rate of approximately 300 min⁻¹ were identified during 3-hour ECG telemetry. On echocardiography, LA diameter (13.3 mm) and transmitral E:A ratio (1.5) were normal (Santilli and Bussadori, 1998; Schober and März, 2005). LV wall thickness and chamber dimensions were unchanged, and LV SF 48 % (Fig. 2A) and LV peak E-to-peak velocity of early diastolic lateral mitral annulus motion (Ea) was mildly elevated (E:Ea, 13.0; reference, 5.0 to 12.0; Schober et al., 2003). A Holter monitor was again recommended and declined; therapy remained unchanged.

The cat was re-evaluated 12 months after the initial exam and had continued to do well with similar telemetric electrocardiographic and echocardiographic findings compared to previous studies with fast runs of narrow complex tachycardia and frequent single VPCs still present. As the cat was not clinically symptomatic and alternative treatments were not readily available the owner decided to continue with the current medication. Other potentially more effective antiarhythmic drugs such as Mexiletine, Atenolol, Amiodarone, Digitalis or combinations of such compounds were not considered due to absence of evidence regarding their antiarrhythmic efficacy in cats and the potential for adverse effects. The cat was not seen again until 14 months later (26 months since initial presentation) and he continued to be overtly asymptomatic. However, cardiac auscultation revealed a fast irregularly irregular rhythm at a rate of 240 min⁻¹, and was otherwise normal. The ECG was most consistent with atrial fibrillation with a rapid ventricular response rate (Fig. 1D) as observed continuously during the 4 hour work-up period although supraventricular tachycardia with slight irregularity could not be completely ruled out. Echocardiography revealed eccentric LV hypertrophy with normal wall thickness and atrial enlargement (LA diameter 19.5 mm; right atrial antero-posterior dimension 22.6 mm, reference range, 10 to 16). There was mild mitral and tricuspid regurgitation, evidence of severe right (subjective) and LV systolic dysfunction (LV SF 15%; Fig. 2B), and severe LV diastolic dysfunction with restrictive transmitral (Peak E 1.62 m/s; reference range, 0.4 to 1.2) and diastolic-dominant pulmonary venous flow. Cardiac rhythm was slightly irregular, and persistent absence of late diastolic transmitral flow, pulmonary venous atrial reversal flow, and late diastolic mitral annulus tissue motion were supportive of presence of atrial fibrillation. Thoracic radiography was consistent with generalized cardiomegaly but absence of congestive heart failure. Cardiac troponin I was mildly elevated at 0.39 ng/mL suggestive of acute or ongoing chronic myocardial injury. A fundic exam was normal as was whole blood taurine concentration



Figure 2: Composite of left ventricular M-mode images recorded from a right-parasternal short axis image at: Panel A) 3 months after initial presentation (LVIDd = 17.1 mm, SF = 48 %); and Panel B) 26 months after initial presentation (LVIDd = 17.5 mm, SF = 15 %). Sweep speed 100 mm/s. LVIDd, left ventricular internal dimension at end-diastole; SF, LV shortening fraction.

(658 nmol/L; reference range, > 200). A serum biochemistry profile was unremarkable, with total T4 within normal limits. The major differentials at that time included tachycardia-induced cardiomyopathy (TIC), ARVC, and unidentified myocardial disease. Due to the severity of cardiac dysfunction and volume overload therapy for advanced heart failure was instituted. This included therapy with furosemide (1 mg/kg q12h, PO; Salix®, Merck Animal Health, Summit, NJ, USA), enalapril (0.22 mg/ kg q12h, PO; Enalapril generic, Woockhard USA LLC, Parsippany, NJ, USA), pimobendan (0.22 mg/kg q12h, PO; Vetmedin®, Boehringer Ingelheim, St Joseph, MO, USA), and clopidogrel (3.3 mg/kg q24h, PO; Clopidogrel generic, Mylan Pharmaceuticals Inc., Morgantown, WV, USA). Concurrently, the dose of sotalol was decreased to 1 mg/kg q12h, PO for three days and 0.5 mg/kg q12h, PO thereafter.

At a recheck 1 week later the cat was reportedly behaving normally. Heart rate was between 200 and 280 min⁻¹, the rhythm was still irregularly irregular, and the remainder of the physical examination unremarkable. Institution of additional antiarrhythmic treatments such as digoxin or diltiazem was declined by the owner.

The cat was represented 13 days later for anorexia, vomiting, and reluctance to move, increased vocalization, and lameness in the right hind limb. Feline arterial thromboembolism or at least severely reduced peripheral blood flow with hind limb ischemia were suspected. Physical examination revealed hypothermia (rectal temperature below 35.6 degrees Celsius; reference range, 38.0 to 39.5), a heart rate of 150 min⁻¹, and moderate dehydration. On cardiac auscultation the rhythm was irregularly irregular and soft heart sounds were present. Femoral pulses were bilaterally present but hypokinetic and variable. Systolic Doppler blood pressure was not attainable. These findings were consistent with cardiogenic shock and a dobutamine constant rate infusion was started at 5 µg/kg/ min and increased to 7 µg/kg/min within 10 min (Dobutamine generic, Hospira Inc., Lake Forest, IL, USA). External heat support was initiated and 0.45 % NaCl solution at maintenance rate (12 mL/h, CRI; 0.45 % sodium chloride solution generic, Baxter Healthcare Corp., Deerfield, IL, USA) administered. After one hour of infusion the systolic blood pressure was measured at 44 mmHg. Blood work revealed a PCV of 0.39 (reference range, 0.25 to 0.46), a total protein of 80 g/dL, severe azotemia (BUN 164 mg/dL and creatinine 7.3 mg/dL; reference ranges, 13 to 30 and 0.9 to 2.1, respectively), hyperphosphatemia (7.3 mg/dL), and increased creatine kinase (1,472 U/L). Due to the grave prognosis, humane euthanasia was performed.

Post mortem examination revealed a normal cardiac weight of 21 g (4.53 g/kg, 0.48 % of body weight; Liu et al., 1981) and grossly visible 4-chamber enlargement, consistent with eccentric remodeling. The LV free wall and interventricular septum each measured 5 mm in thickness, and the right ventricular free wall was 2 mm thick. Histo-

logical examination of 11 myocardial samples including the free wall of left and right atrium and left and right ventricle, LV papillary muscles, left and right auricles, and interventricular septum was performed. Tissue was processed by routine methods, embedded in paraffin, sectioned at a thickness of 3 µm, stained with hematoxylin and eosin (H & E) as well as Masson's Trichrome, and qualitatively evaluated. To assess presence of apoptosis of cardiomyocytes, immunohistochemistry was performed, staining with antibody against cleaved caspase 3. There was mild fiber atrophy, mild multifocal endocardial and subendocardial fibrosis, and rare intramural coronary arterial narrowing in segments from the interventricular septum, the LV free wall, and the papillary muscles. However, the vast majority of the sections appeared normal (Fig. 3 A-F). Rare, scattered "wavy fibers" (Bouchardy and Majno, 1974 and Tidholm et al., 1998) were found across the right ventricular free wall. The myocardium of the atria and auricles was unremarkable. There was no evidence of necrosis, acute degeneration, inflamma-



Figure 3: Composite of histopathologic findings from different sections of the heart. Panels A and B (right ventricular free wall), panels C and D (left ventricular free wall), and panels E and F (interventricular septum). Normal appearance of myofibers (panels A, C, E; H & E stain) with minimal interstitial fibrosis (fibers stained blue; panels B, D, F; Masson's Trichrome stain). All images: 20 x magnification, bar = 50μ m.

tion, fiber disarray, or fatty infiltration on H & E stained slides, and immunohistochemistry did only identify very minimal apoptosis (few scattered single cells). Mild pleural effusion was present. Considering historical, clinical, electrocardiographic, echocardiographic, and pathologic findings a final diagnosis of TIC was made but the origin of the underlying arrhythmia remained unexplained.

Discussion

Tachycardia-induced cardiomyopathy or tachycardiomyopathy is a potentially reversible form of ventricular remodeling and dysfunction secondary to chronic supraventricular and ventricular tachyarrhythmias (Fenelon et al., 1996; Shinbane et al., 1997; Walker et al., 2004; Khasnis et al, 2005). TIC has been reported in experimental rapid pacing models and secondary to naturally-acquired tachyarrhythmia in dogs (Whipple et al., 1962; Shinbane et al., 1997; Wright et al., 1999; Walker et al., 2004; Khasnis et al., 2005; Foster et al., 2006) and other species. Moreover, repetitive ventricular premature beats or paroxysms of any type of tachycardia affecting more than 15% of the daily heart beats may result in TIC in people, although the latter may need several months or even years to develop (Fenelon et al., 1996; Yarlagadda et al., 2005; Watanabe et al., 2008). TIC can be a primary entity, where the tachycardia alone induces cardiac pathology; or can be a secondary complicating event in the presence of underlying structural heart disease (Fenelon et al., 1996).

The diagnosis of TIC is based on strong clinical suspicion if chronic tachyarrhythmia is identified and primary or secondary cardiac disease causing a similar final phenotype is ruled out. By definition (Packer et al., 1986) demonstration of normal cardiac chamber function before the onset of cardiac impairment and subsequent documentation by serial evaluations of progressive functional deterioration after frequent and continuous tachycardia is needed for diagnosis, as observed in our cat. Moreover, the correct diagnosis should be confirmed by demonstration of improvement of cardiac function after cure or control of the arrhythmia (Yarlagadda et al., 2005). However, depending on severity and duration of the arrhythmia functional recovery may be complete, partial, or totally absent (Packer et al., 1986). In the present case, progression to the point of heart failure rather than improvement occurred despite antiarrhythmic therapy, likely because treatment did not control the arrhythmia. To the author's knowledge, the causative relationship between tachycardia and LV remodeling was first hypothesized in 1913 in a man diagnosed with atrial fibrillation who had no underlying structural heart disease (Gossage et al., 1913). In 1949, Phillips and Levine described the association between atrial fibrillation and a reversible form of heart failure. Whipple et al (1962) reported on the first experimentally-induced TIC resulting in congestive heart failure due to rapid atrial pacing. Since that time animal

models of rapid-pacing induced cardiomyopathy have been established and are commonly used to create and study biventricular heart failure (Shinbane et al., 1997). Typically, a ventricular pacing rate of 240 min⁻¹ is used and reliably results in low output heart failure within 3 to 4 weeks (Fenelon et al., 1996).

Reversal of tachycardia-induced remodeling and chamber dysfunction and resolution of clinical signs have been documented in both humans (Fenelon et al., 1996, Shinbane et al., 1997) and dogs (Wright et al., 1999; Foster et al, 2006) after control of fast heart rate or correction of abnormal rhythm, although the outcome may be quite variable. Systolic dysfunction that occurs with rapid pacing-induced cardiomyopathy may reverse as quickly as two weeks after pacing is discontinued. However, in people with naturallyacquired TIC there is often an initial rapid improvement after about one month, followed by a more gradual improvement that reaches a maximum after six to eight months (Fenelon et al., 1996). A noticeable increase in systolic chamber function was reported within two days of ablation of bypass tract tachycardia in three dogs, with continued improvement over the next three to 14 months (Wright et al., 1999). The natural history of TIC is dependent upon the rate and duration of the arrhythmia (Khasnis et al., 2005) as well as the potentially underlying disease process. Once an advanced state of myocardial remodeling has developed reversibility may not be observed. If return to normal cardiac function after abolishment of the arrhythmia cannot be documented, there are a number of histopathologic findings that make the distinction of TIC from other types of myocardial disease possible. In general, TIC is associated with only mild and often non-specific myocardial changes (Tab. 1; Packer et al., 1986). Absence of more than trivial myocardial hypertrophy; myofiber disarray; diffuse multifocal endocardial, subendocardial, perivascular, or interstitial fibrosis; inflammation; and fatty or fibro-fatty infiltration makes the presence of hypertrophic, restrictive, dilated, or arrhythmogenic right ventricular cardiomyopathy as well as Uhl's anomaly and myocarditis unlikely. Other, undetermined types of cardiomyopathy would still be potential differentials; however, their clinical and histopathologic characteristics are poorly understood (Ferasin et al., 2003; Cote et al., 2004; Glaus and Wess, 2010). The clinical progression of the cat of this report, including normal cardiac anatomy and function at baseline, the history of chronic paroxysmal tachyarrhythmia that progressed to atrial fibrillation, and progressive chamber remodeling and dysfunction were all consistent with TIC. The mild histological myocardial changes seen in this cat are similar to that reported in TIC in people (Tab. 1, Packer et al., 1986). The cellular and molecular mechanisms leading to TIC are incompletely understood. Proposed mechanisms include abnormal cardiomyocyte calcium handling leading to calcium overload and associated upregulated apotosis, cell atrophy, and necrosis; ischemia with energy starvation and mitochondrial injury; down regulation of myocyte beta-1 receptors; oxidative stress; and reduced myocardial

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blood flow reserve (Fenelon et al., 1996 and Shinbane et al., 1997). To the authors' knowledge this is the first report of naturally-acquired TIC in a cat. Due to the detrimental nature of uncontrolled arrhythmias with fast heart rate, all attempts should be undertaken to optimize heart rate and normalize cardiac rhythm in cats with "idiopathic" or secondary tachyarrhythmias. Ideally, the cause of the arrhythmia is identified and eliminated (e.g., ablation of an accessory pathway), the arrhythmogenic substrate improved (e.g., elimination of myocardial ischemia or reduction of fibrosis), or trigger events abolished (e.g., electrolyte imbalanced corrected or exaggerated sympathetic stimulation antagonized). At any rate, if tachycardia control is not accomplished, the development of TIC with poor prognosis is to be expected.

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	People	Cat
LV dimension	Increased	Increased
LV systolic function	Decreased	Decreased
LV diastolic function	Decreased	Decreased
LV Wall thickness	Normal to reduced	Normal to reduced
Mitral regurgitation	Mild	Mild
Myocardial fibrosis	Mild	Mild
Myocardial fiber lengthening	Mild to moderate	Mild
Myocyte atrophy and loss	Mild to moderate	Mild
Fatty infiltration	Absent	Absent
Myocardial inflammation	Absent	Absent
Myocardial apoptosis	Mild	Minimal

LV, left ventricular.

References

Bouchardy B., Majno G.: Histopathology of early myocardial infarcts. A new approach. Am. J. Pathol. 1974, 74: 301–330.

Cote E., Manning A. M., Emerson D. et al.: Assessment of the prevalence of heart murmurs in overtly healthy cats. J. Am. Vet. Med. Assoc. 2004, 225: 384–388.

Fenelon G., Wijns W., Andries E. et al.: Tachycardiomyopathy: mechanisms and clinical implications. Pacing Clin. Electro-physiol. 1996, 19: 95–106.

Ferasin L., Sturgess C. P., Cannon M. J. et al.: Feline idiopathic cardiomyopathy: a retrospective study of 106 cats (1994–2001). J. Fel. Med. Surg. 2003, 5: 151–159.

Foster S. F., Hunt G. B., Thomas S. P. et al.: Tachycardia-induced cardiomyopathy in a young Boxer dog with supraventricular tachycardia due to an accessory pathway. Aust. Vet. J. 2006, 84: 326–331.

Glaus T., Wess G.: Left ventricular hypertrophy in the cat – "when hypertrophic cardiomyopathy is not hypertrophic cardiomyopathy". Schweiz. Arch. Tierheilkd. 2010, 152: 325–330.

Gossage A. M., Braxton Hicks J. A.: On auricular fibrillation. Q. J. Med. 1913, 6: 435–440.

Khasnis A., Jongnarangsin K., Abela G. et al.: Tachycardia-induced cardiomyopathy: a review of literature. Pacing Clin. Electrophysiol. 2005, 28: 710–721.

Liu S. K., Maron B. J., Tilley L. P.: Feline hypertrophic cardiomyopathy: gross anatomic and quantitative histologic features. Am. J. Pathol. 1981, 102: 388–395. *Packer D. L., Bardy G. H., Worley S. J. et al.*: Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. Am. J. Cardiol. 1986, 57: 563–570.

Phillips E., Levine S. A.: Auricular fibrillation without other evidence of heart disease; a cause of reversible heart failure. Am. J. Med. 1949, 7: 478–489.

Santilli R. A., Bussadori C.: Doppler echocardiographic study of left ventricular diastole in non-anaesthetized healthy cats. Vet. J. 1998, 156: 203–215.

Schober K. E., Fuentes V. L., Bonagura J. D.: Comparison between invasive hemodynamic measurements and noninvasive assessment of left ventricular diastolic function by use of Doppler echocardiography in healthy anesthetized cats. Am. J. Vet. Res. 2003, 64: 93–103.

Schober K. E., Maerz I.: Doppler echocardiographic assessment of left atrial appendage flow velocities in normal cats. J. Vet. Cardiol. 2005, 7: 15–25.

Shinbane J. S., Wood M. A., Jensen D. N. et al.: Tachycardiainduced cardiomyopathy: a review of animal models and clinical studies. J. Am. Coll. Cardiol. 1997, 29: 709–715.

Tidholm A., Haggstrom J., Jonsson L.: Prevalence of attenuated wavy fibers in myocardium of dogs with dilated cardiomyopathy. J. Am. Vet. Med. Assoc. 1998, 212: 1732–1734.

Walker N. L., Cobbe S. M., Birnie D. H.: Tachycardiomyopathy: a diagnosis not to be missed. Heart 2004, 90: e7.

Watanabe H., Okamura K., Chinushi M. et al.: Clinical characteristics, treatment, and outcome of tachycardia induced cardiomyopathy. Int. Heart J. 2008, 49: 39–47.

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Whipple G. H., Sheffield L. T., Woodman E. G. et al.: Reversible congestive heart failure due to chronic rapid stimulation of the normal heart. Proc. N. Engl. Cardiovasc. Soc. 1962, 20: 39–40.

Wright K. N., Mehdirad A. A., Giacobbe P. et al.: Radiofrequency catheter ablation of atrioventricular accessory pathways in 3 dogs with subsequent resolution of tachycardia-induced cardiomyopathy. J. Vet. Intern. Med. 1999, 13: 361–371.

Yarlagadda R. K., Iwai S., Stein K. M. et al.: Reversal of cardiomyopathy in patients with repetitive monomorphic ventricular ectopy originating from the right ventricular outflow tract. Circulation 2005, 112: 1092–1097.

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