Electrocardiography-documented sudden cardiac death due to ventricular fibrillation in a young cat without echocardiographic evidence of severe structural heart disease

Elektrocardiografie-gedocumenteerde plotse cardiale dood ten gevolge van ventriculaire fibrillatie bij een jonge kat zonder echocardiografische tekenen van ernstige structurele hartziekte

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ABSTRACT

An asymptomatic three-year-old male European Domestic Shorthair cat was referred for an irregular heart auscultation. Apart from localized concentric left ventricular septal hypertrophy, echocardiography was normal. However, ventricular ectopy was observed on electrocardiography (ECG) during echocardiography. The cat died suddenly 55 minutes after the start of the subsequent Holter ECG. Analysis showed sinus rhythm interrupted by 192 single ventricular premature complexes, a ventricular couplet, two incidents of ventricular bigeminy and two runs of ventricular tachycardia (VT). The last run of VT deteriorated into ventricular fibrillation (VF) and ultimately cardiac arrest. Structural heart disease including mild feline hypertrophic cardiomyopathy or myocarditis, primary arrhythmia (channelopathy) or extra-cardiac disease were maintained as differential diagnosis. Unfortunately, a necropsy was declined leaving no definitive diagnosis available. In this case report, it is shown that VF due to ventricular ectopy is a possible cause of SCD even in asymptomatic cats with only mild echocardiographic signs of structural heart disease.

SAMENVATTING

Een asymptomatische, drie jaar oude, mannelijke Europese korthaar werd doorverwezen voor een onregelmatige hartauscultatie. Behalve gelokaliseerde concentrische links ventriculaire septumhypertrofie was de echocardiografie normaal. Tijdens het echocardiografisch onderzoek werd echter ventriculaire ectopie waargenomen op de elektrocardiografie (ECG). De kat overleed plotseling 55 minuten na het begin van het vervolgens uitgevoerde Holter ECG. Analyse toonde een sinusritme onderbroken door 192 enkelvoudige ventriculaire premature complexen, een ventriculair couplet, twee incidenten van ventriculaire bigeminus en tenslotte twee “runs” van ventriculaire tachycardie (VT). De laatste run ging over in ventrikelfibrillatie (VF) en uiteindelijk hartstilstand. Helaas werd een lijkschouwing afgewezen, waardoor geen definitieve diagnose kon worden gesteld. In deze casereport wordt aangetoond dat VF als gevolg van ventriculaire ectopie een mogelijke oorzaak is van plotse cardiale sterfte bij asymptomatische katten met slechts milde echocardiografische tekenen van een structurele hartziekte.

INTRODUCTION

Ventricular arrhythmia (VA) is commonly observed in cats with structural heart disease (MacDonald et al., 2011). According to one retrospective study, 96% of the cats presenting with VA have concurrent echocardiographic abnormalities (Côté and Jaeger, 2008). Feline cardiomyopathies associated with ventricular arrhythmias include hypertrophic (HCM), restrictive, unclassified and dilated cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy (MacDonald et al., 2011). Other important cardiac causes of VA include congenital or valvular heart disease, cardiac neoplasia, infectious myocarditis and endocarditis (Fox et al., 2000; Côté and Jaeger, 2008; MacDonald et al., 2011; Anderson, 2018). In addition, a variety of extracardiac causes have been associated with VA in cats. These include electrolyte imbalances, hyperthyroidism, severe anemia, hypovolemia, hypoxia, intra-abdominal disease and alterations in sympathetic tone (Peterson et al., 1982; Love et al., 2010; MacDonald et al., 2011; Anderson, 2018; Bartoszuk et al., 2019). A low number of ventricular premature complexes (VPCs) can also be found in healthy cats and their frequency appears to increase with increasing age (Ware, 1999; Hanås et al., 2009; Jackson et al., 2014). Depending on the study, a median of 3 VPCs (range 0-146) (Hanås et al., 2009) or mean of 4 VPCs per 24 hours (Jackson et al., 2014) can be considered normal in cats in the home environment.

In this case report, a young European Domestic Shorthair cat is described without clinical signs nor echocardiographic evidence of severe structural heart disease, in which ventricular tachycardia (VT) leading to ventricular fibrillation (VF) and sudden cardiac death (SCD) is documented by Holter electrocardiography (ECG).

CASE DESCRIPTION

A three-year-old, male, neutered European Domestic Shorthair of 6.7 kg (body condition score 6/9) was referred to the Ghent University Small Animal Teaching Hospital because of an incidentally detected irregularity on heart auscultation six weeks earlier.

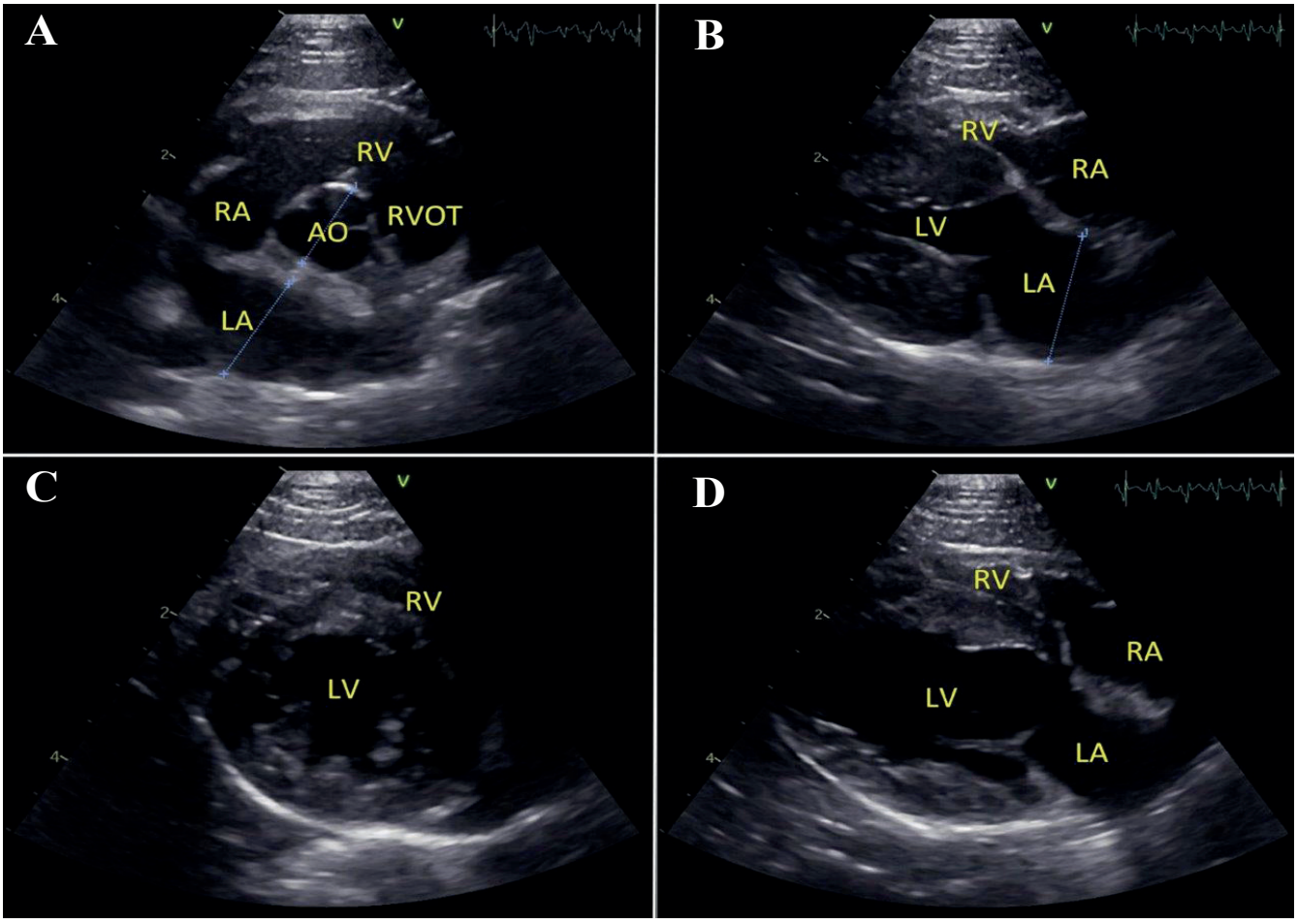

Figure 1. Two-dimensional echocardiographic images of the cat. A. Right parasternal short axis view at the level of the aortic valve during early-diastole. B. Right parasternal long axis view during end-systole, focused on the left atrium. C. Right parasternal short axis view at the level of the papillary muscle during end-diastole. D. Right parasternal long axis view during end-diastole. Normal left and right atrial size was appreciated (A and B). Left ventricular septal and free wall thickness in end-diastole were just above the upper reference limit (C and D) and classified equivocal (Häggström et al., 2016). Local hypertrophy was present D. A detailed summary of all echocardiographic measurements can be found in Table 1.
AO: aorta; RA: right atrium; RV: right ventricle; RVOT: right ventricular outflow tract; LA: left atrium; LV: left ventricle.

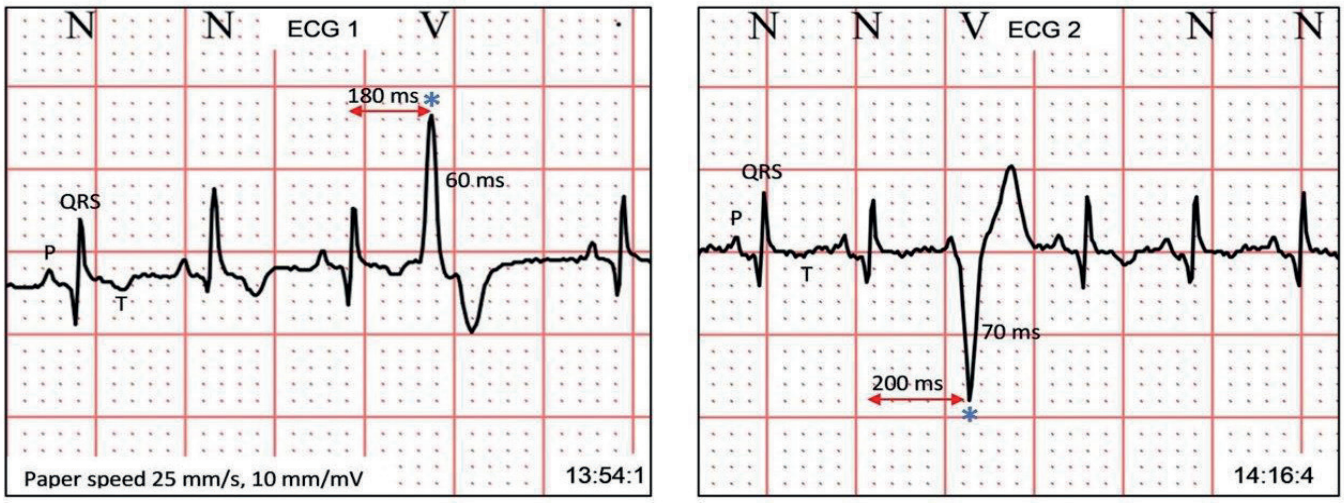

Figure 2. Strip from the 55 min Holter electrocardiography (ECG) recording. ECG 1 shows sinus rhythm interrupted by a wide QRS complex (>40 ms) (blue asterisk) with a short coupling interval (180 ms or 330 beats per minute), not preceded by a P wave and followed by a compensatory pause. This is compatible with a ventricular premature complex (VPC). ECG 2 starts again with sinus rhythm interrupted by another wide QRS complex (blue asterisk) with a slightly longer coupling interval, longer QRS duration and different morphology, again compatible with a VPC.

Except for sporadic retching behavior and a conservatively treated mild rectal mucosal protrusion one year prior to presentation, no relevant medical history or clinical signs were noted. At the time of referral, the cat did not display any clinical signs according to the owner. On the day of consultation, the cat was alert. Heart auscultation revealed an irregular rhythm in absence of a murmur or pulse deficit. The remaining physical examination was unremarkable.

Localized concentric left ventricular septal hypertrophy and left ventricular free wall thickness within the equivocal range (0.54 cm) (Häggström et al., 2016; Chetboul et al., 2006; Kittleson and Côté, 2021) were the only abnormalities identified on echocardiography (Vivid E95, GE Healthcare, Diegem, Belgium) (Figure 1). A summary of the echocardiographic measurements is provided in Table 1. Ventricular ectopy including VPCs and runs of monomorphic VT (up to 200 beats-per-minute (bpm)) were noticed on single lead ECG during the ultrasound examination. A six-lead ECG was attempted but tracings were of poor quality due to movement of the patient. Bloodsamples were sent to the lab, but the complete intended analysis (biochemistry, hematology, electrolytes, cardiac troponin I (cTnI) and *Toxoplasma gondii* antibodies) was initially not performed considering the cat died during the further course of the investigations. In order to further assess the severity and prevalence of the observed arrhythmias, the cat was hospitalized for Holter ECG (Vista Plus, Novacor, Rueil-Malmaison, France) monitoring. However, 55 minutes after Holter ECG attachment, the cat went into cardiopulmonary arrest without any observed prior clinical signs. Analysis of the available 55 minutes Holter ECG recording revealed sinus rhythm (between 170 to 240 beats per minute (bpm)), 192 single VPCs with both right and left bundle branch block morphology (Figure 2), one ventricular couplet (coupling interval 160 ms), two incidents of bigeminy and finally, two runs of VT occurring approximately one minute prior to SCD. The first run of VT lasted five monomorphic QRS complexes and displayed a VT rate between 240 and 286 bpm. The last run of VT deteriorated into VF and ultimately cardiac arrest. This run consisted of nine polymorphic QRS complexes with a VT rate between 400 and 750 bpm displaying R-on-T phenomenon (Figure 3). After degeneration into VF, four morphologically different stages were identified based on the description of Wiggers (1940), before the onset of asystole (Wiggers, 1940; Aras et al., 2017) (Figure 4).

Cardiopulmonary resuscitation (CPR) was initiated according to RECOVER guidelines after the cat was found in the state of cardiopulmonary arrest, but was unsuccessful (Fletcher et al., 2012). Necropsy was offered because it could provide valuable insights into the cause of this cat's arrhythmia. Unfortunately, the offer was declined by the owner which is an important limitation to this case report. For educational purposes, cTnI and *Toxoplasma gondii* antibodies (IgM and IgG) were measured on the available blood sample to exclude (*Toxoplasma)* myocarditis. Cardiac troponin I was within normal limits (cTnI 0.035 µg/l; reference interval: 0.001–0.0373 µg/l) and IgM and IgG were non-indicative for recent *T gondii* infection (Sleeper et al., 2001).

DISCUSSION

In this case report, SCD is described in a young cat related to ventricular arrhythmias without echocardiographic evidence of severe structural heart disease.

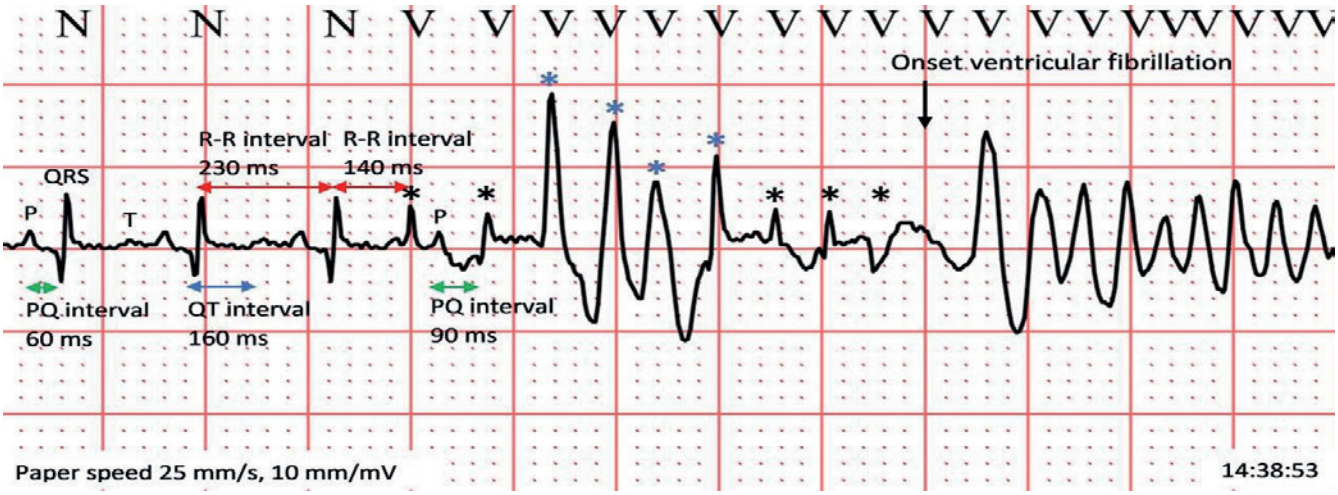

Figure 3. Strip from the 55 minutes Holter electrocardiography (ECG) recording. The ECG trace starts with a regular narrow QRS rhythm (± 40 ms, three QRS complexes) with a rate of approximately 260 beats per minute (bpm). Clear P waves precede each of the first three QRS complexes, compatible with sinus rhythm. Subsequently, an irregular rhythm is observable (nine QRS complexes indicated by an asterisk) (400-750 bpm) displaying clear AV dissociation. Whilst complex 6, 7, 12 and 13 (black asterisk) have a QRS duration just above >40 ms, complex 8, 9, 10 and 11 (blue asterisk) present with a different morphology characterized by a marked QRS prolongation and increased amplitude. This rhythm is compatible with ventricular tachycardia. The last ventricular complex (black asterisk) has a very short coupling interval of 80 ms (or 750 bpm) and occurs near ventricular repolarization, after which the rhythm deteriorates into ventricular fibrillation (also visible in Figure 4). This rhythm is characterized by fast, wide and irregular undulations of the baseline without clear QRS-T morphology, here with a rate of approximately 750 bpm (Mavropoulou, 2018). No QT prolongation was observed prior to the onset of ventricular fibrillation.

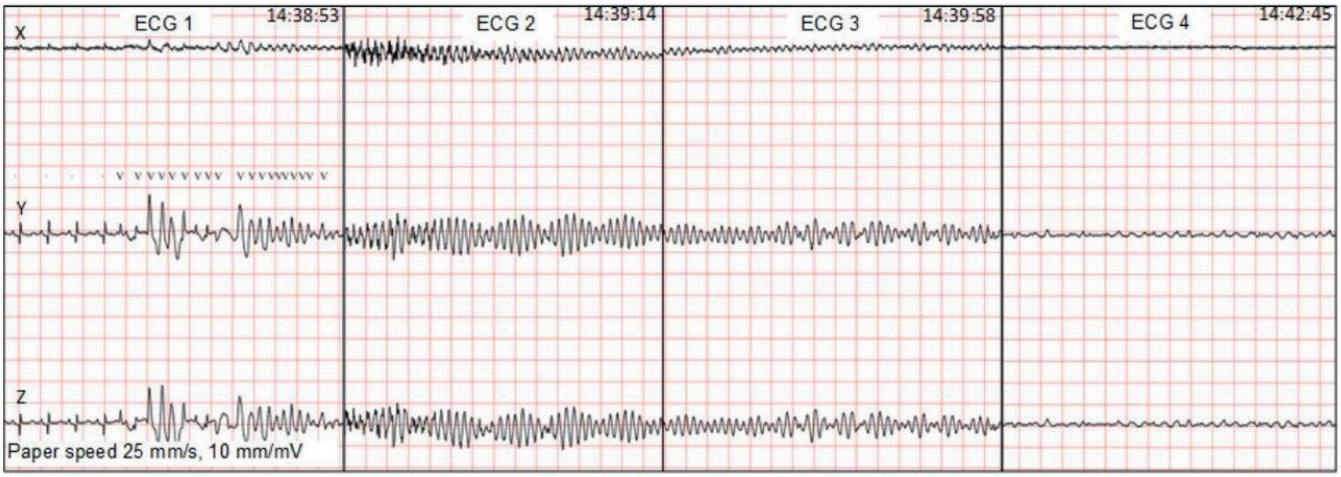

Figure 4. The electrocardiography (ECG) traces show the four phases of ventricular fibrillation (VF) according to Wiggers (1940). ECG 1: Onset of VF characterized by several fast and wide ventricular deflections at a rate of approximately 750 beats per minute (bpm), consistent with stage 1 or the tachysystolic or undulatory phase. Usually, this phase lasts only a few seconds in humans. ECG 2: Very fast, wide deflections appear with an irregular amplitude at a rate between 750-1000 bpm, consistent with stage 2 or convulsive incoordination. Usually, this phase lasts less than 40 seconds as it reflects increasingly smaller zones of myocardial contraction. The trace mimics the appearance of Torsade De Pointes; however, the latter should be associated with a prolonged QT interval which was not present in the cat. ECG 3: fast, but more irregular wide deflections of decreasing amplitude at a rate of 600-750 bpm. This is consistent with stage 3 or tremulous incoordination and was observed about 30 seconds after VF onset. ECG 4: small, irregular deflections consistent with atonic fibrillation appearing >3 min after the onset of VF (Aras et al., 2017; Mavropoulou, 2018).

Characteristics of spontaneous arrhythmias leading to SCD have been described in dogs, but no data are available in cats (Santilli et al., 2021). In the current case, both the arrhythmia leading to SCD as well as the heart rhythm preceding SCD were documented on ECG.

The timing of the first occurrence of VA prior to SCD was unknown in this cat. Based on the incidental finding of an arrhythmia on auscultation during a routine check-up by the referring veterinarian six weeks earlier, it was hypothesized that an arrhythmia had been present for several weeks prior to referral. Conversely, if this cat had severe tachyarrhythmia with a daily arrhythmia load >15%, it could be argued

Table 1. Summary of two-dimensional (2D) and Doppler echocardiographic measurements.

Two-dimensional variable	Measurement	Reported 95% prediction interval*
Interventricular septum thickness in diastole	0.52 cm	0.31 – 0.54 cm
Left ventricular internal diameter end diastole	1.55 cm	1.37 – 2.14 cm
Left ventricular posterior wall thickness in diastole	0.54 cm	0.31 – 0.54 cm
Interventricular septum thickness in systole	0.80 cm	0.47 – 0.93 cm
Left ventricular internal diameter end systole	0.59 cm	0.61 – 1.41 cm
Left ventricular posterior wall thickness in systole	1.08 cm	0.51 – 0.94 cm
Septal bulge	0.62 cm	
% Fractional shortening	62%	28 – 62 %
Aorta diameter	0.93 cm	0.81 – 1.29 cm
Left atrial diameter	1.50 cm	0.89 – 1.58 cm
Left atrium to aorta ratio	1.41	0.89 – 1.44
Right atrial diameter	1.45 cm	
Doppler variable	**Measurement**	**Reported mean ± standard deviation or reference range****
Mitral valve EA velocity fused	82.0 cm/s	100 ± 10 cm/s
Aortic valve velocity maximum	1.11 m/s	0.7 – 1.4 m/s
E' lateral (mitral valve annulus) (tissue Doppler)	10.13 cm/s	1.8 – 8.7 cm/s
A' lateral	5.98 cm/s	0.0 – 5.5 cm/s
S' lateral	10.48 cm/s	1.9 – 5.9 cm/s
Isovolumic relaxation time (transmitral)	48.44 ms	33 – 82 ms

* According to Häggström et al. (2016) based on 19 866 healthy pure-bred cats. The displayed results were selected based on an ideal body weight of 6.0 kg (current weight 6.7 kg, body condition score 6/9).
** According to Chetboul et al. (2006) based on 31 Domestic Shorthair cats with a mean bodyweight of 4.0 ±1.2 kg

that the cat possibly exhibited tachycardia-induced cardiomyopathy, as observed in humans (Fenelon et al., 1996; Greet et al., 2020). Eight and ten months earlier, respectively, the cat was seen by the referring veterinarian and our clinic for a rectal prolapse, and no auscultatory abnormalities were reported. No clinical signs such as exercise intolerance, tachypnea, dyspnea, weakness or syncope had been witnessed by the owner.

The cat in the current report displayed VA in the immediate period before and leading to SCD. The VA displayed malignancy criteria, such as polymorphism, a fast instantaneous VPC rate (>300 bpm), the presence of bigeminy, couplets, triplets and two fast runs of VT before the onset of VF and cardiac arrest. Unfortunately in cats, evidence relating VA load and grading to SCD is lacking. Therefore, predicting the risk of SCD based on the presence of VA or any of its ECG characteristics in an individual cat is unreliable. In the cat of the present report, a run of VT with R-on-T phenomenon deteriorating into VF, was the documented cause of SCD (Figure 3).

In the vast majority of cats, both VA and SCD are related to structural heart disease, most notably HCM (Côté and Jaeger, 2008; Wilkie et al., 2015; Santilli et al., 2021). However, the exact relationship between VA, SCD and HCM needs further study. In a cohort of 255 cats with HCM, SCD was the least frequent cause of mortality (4.7%) following congestive heart failure (17.3%) and arterial thromboembolism (9.0%), and the presence of arrhythmia on auscultation was associated with an increased risk for SCD. However, the exact cause of SCD was unknown in most cats and arrhythmia (VF) causing SCD was only confirmed once (Payne et al., 2015). Unfortunately, data on the prevalence of VA and SCD in the general cat population, including cats without severe structural heart disease is unknown and likely underreported (Payne et al., 2015). The cat in the current report had no echocardiographic evidence of severe structural heart disease apart from localized concentric left ventricular septal hypertrophy and diastolic left ventricular free wall measurements within the equivocal range (Häggström et al., 2015; Häggström et al., 2016; Kittleson and Côté, 2021). Perhaps the echocardiographic findings in the present report could represent mild HCM (American College of Veterinary Internal Medicine (ACVIM) stage B1) (Luis Fuentes et al., 2020). Unfortunately, post-mortem diagnosis would require necropsy and histopathology, which were not performed.

Another plausible differential diagnosis for local ventricular hypertrophy, VA and SCD in this cat would be infectious myocarditis (Liu, 1985; Wilkie et al., 2015; Santilli et al., 2021). Given the time period between the onset of VA and the cTnI measurement, myocarditis remains a possible differential diagnosis despite normal cTnI values. In humans with myocarditis, cTnI only has a sensitivity of 34% (Smith et al., 1997). Several infectious agents in cats have been de-

scribed in association with myocarditis and on some occasions, supra- or ventricular tachyarrhythmia or conduction abnormalities such as atrioventricular block have been reported. These include *Toxoplasma gondii*, *Bartonella spp*, *Borrelia burgdorferi*, *Feline coronavirus*, *Salmonella typhimurium*, *Feline immunodeficiency virus* and very recently *severe acute respiratory syndrome coronavirus 2* or SARS-CoV-2 (Simpson et al., 2005; Nakamura et al., 2011; Rolim et al., 2016; Donovan et al., 2018; Joseph et al., 2018; Ernandes et al., 2019; Vercelli et al., 2019; Tørnqvist-Johnsen et al., 2020; Chetboul et al., 2021; Ferasin et al., 2021). Unfortunately given the sudden death of the patient during the investigations, no extensive infectious testing was performed. *Toxoplasma gondii* IgM and IgG antibody testing was negative. Other tests were not performed.

A last differential diagnosis for VA and SCD could be a primary congenital arrhythmia or channelopathy. The latter has been described in humans and dogs but not in cats, although their existence is plausible (Moise et al., 1994; Ware et al., 2015; Meurs et al., 2016; Wiberg et al., 2020; Brugada-Terradellas et al., 2021).

Other non-cardiac causes for VA and thereby potentially SCD, include electrolyte abnormalities, anemia, intra-abdominal disorders and diseases that alter sympathetic tone (MacDonald et al., 2011; Anderson, 2018; Bartoszuk et al., 2019). In the current case, none of them could be excluded.

Unlike in dogs, ambulatory Holter ECG monitoring in cats is much more often performed in-clinic than at home (Abbott, 2005; Bartoszuk et al., 2019; Ferasin et al., 2020). Some cardiologists/clinicians consider in-clinic Holter ECG monitoring safer for the cat than at home Holter ECG monitoring (i.e. lower risk of getting caught or entangled in the cables, especially for outdoor cats) (Ferasin et al., 2020). However, others have questioned the safety and validity of such approach as this may result in a higher sympathetic tone, which could potentially worsen pre-existing arrhythmias and may not represent the actual arrhythmia burden (Abbott, 2005; Hanås et al., 2017; Ferasin et al., 2020; Bartoszuk et al., 2019). In the cat of the present case, several measures were taken to minimize stress during device attachment and ECG recording. For example, the cat was hospitalized in a separate, dedicated, cat-friendly hospitalization unit with individual housing. Additional measures to avoid stress in the hospitalization environment included the use of pheromones (Feliway Optimum, CEVA SANTÉ ANIMALE, France (from: https://www.feliway.com/nl/producten/feliway-optimum-verdamper), a prohibition of phone calls (in the cat hospitalization) and soft music. Nevertheless, the authors cannot exclude any detrimental effect of stress on the VA present in this cat. Alternative ECG monitoring techniques such as implantable loop recorders or adhesive patch recorders could be considered but carry a significant cost and may not always provide a similar quantitative analysis (Willis, 2018; Ogawa et al., 2022).

The cat in this study was not on antiarrhythmic treatment during Holter ECG monitoring following the initial consultation. The rationale is that not every VA requires antiarrhythmic treatment and that a Holter ECG is the most reliable way to estimate the need for antiarrhythmic treatment in dogs and cats (Willis, 2018). In addition, performing a baseline Holter allows clinicians to evaluate any positive or negative effect on the arrythmia burden (da Silva, 2018). Given the oral pharmacokinetics of antiarrhythmic drugs such as sotalol and amiodarone (i.e. peak plasma level sotalol two to four hours after oral intake in humans) and the onset of SCD 55 minutes after attaching the Holter ECG, it is debatable whether oral administration of antiarrhythmic drugs prior to Holter ECG monitoring could have potentially reduced VA burden, or prevented SCD during Holter in this cat (Hanyok, 1993). Besides, whilst antiarrhythmic drugs have been shown to reduce VA burden in some cases, studies in veterinary medicine have generally failed to show any reduction in the occurrence of SCD (MacDonald et al., 2011; da Silva, 2018). A continuous rate infusion of lidocaine or esmolol might have reduced VA frequency during Holter ECG (Verschoor-Kirss et al., 2022). However, this is not routinely performed during Holter ECG and was not considered due to the absence of clinical symptoms at presentation, the relatively low VT rate (up to 200 bpm) during echocardiography, and the perceived benefit of having a baseline Holter ECG. Treatment of VF consists of immediate identification of loss of consciousness and pulses and direct initiation of cardiopulmonary resuscitation including electrical cardioversion. Thus far, successful management of this rhythm in cats in a clinical setting has only been described once (Berlin et al., 2020). Unfortunately, at the time of initiation of cardiopulmonary resuscitation in the present case, asystole was noted on ECG.

CONCLUSIONS

In this report, a rare case of Holter ECG-documented spontaneous sudden arrhythmic death in a young cat without echocardiographic evidence of severe structural heart disease is described. The occurrence of a run of VT with R-on-T phenomenon degenerating into VF, was the event leading to death. Structural heart disease, such as mild hypertrophic cardiomyopathy (ACVIM stage B1) or myocarditis, primary arrhythmia (channelopathy) or extracardiac systemic disease were regarded as possible causes of the ventricular ectopy and SCD in this cat. Unfortunately, the lack of a necropsy or histopathology limits further speculation on the etiology of the arrhythmia in this cat. Nevertheless, in this case report, it is shown that VF due to VA is a potential cause of SCD

in asymptomatic cats, even in the absence of echocardiographic evidence of severe structural heart disease.

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