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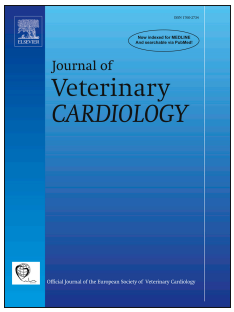

Supraventricular tachycardia in 23 cats; comparison with 21 cats with atrial fibrillation (2004-2014)

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Supraventricular tachycardia in 23 cats; comparison with 21 cats with atrial fibrillation (2004-2014)

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Abstract

Introduction: Supraventricular tachycardia (SVT) has not been well-described in cats. The aim of this study was to describe the signalment, clinical findings and outcome for cats with SVT versus cats with atrial fibrillation (AF).

Animals: Forty-four client-owned cats; 23 cats with SVT and 21 with AF

Methods: Retrospective study. Clinical characteristics were compared between groups using a two-sample t-test or Mann-Whitney U test. Kaplan-Meier survival curves were generated to assess for impact of rhythm diagnosis, presence of ventricular arrhythmia, left atrial diameter, heart rate (HR) and congestive heart failure (CHF) status on cardiac death. Differences in survival between groups were compared using Mantel-Cox logrank comparison of Kaplan-Meier survival curves.

Results: Cats with supraventricular arrhythmias most commonly presented with respiratory distress (10 of 44 cats). Cats with AF had a slower median HR (220 [range 180-260 beats per minute (bpm)] compared to cats with SVT (300 [range 150-380] bpm, $p<0.001$). All cats with AF had structural heart disease whereas 4 cats with SVT had no structural abnormalities. Left atrial diameter was significantly larger in cats with AF (23.7(16.2-40.1) mm, compared to 19.1 (12.8-31.4) mm in SVT cats; $p=0.02$)). Median survival was 58 days [1–780] in cats with AF, and 259 days (2 - 2295] in cats with SVT ($p=0.1$). Cats with signs of CHF had a shorter survival time ($p=0.001$).

Conclusions: Most cats with AF or SVT have advanced structural heart disease. Some cats with SVT had structurally normal hearts, suggesting that SVT in cats is not always a consequence of atrial enlargement.

Keywords:

Feline

Arrhythmia

Survival

Abbreviation table	
AF	atrial fibrillation
ATE	arterial thromboembolism
CHF	congestive heart failure
LA	left atrium
LAD	left atrial diameter
LAE	left atrial enlargement
LV FS%	left ventricular fractional shortening
LVID	left ventricular internal diameter
SCD	sudden cardiac death
SVT	supraventricular tachycardia
VSD	ventricular septal defect

Introduction

Supraventricular tachycardia can be defined as any rapid rhythm originating from the SA node, atrial myocardium, atrioventricular node/junction, or great vessels connecting to the atria (venae cavae, pulmonary veins, coronary vein) [1]. The arrhythmia may arise due to spontaneous depolarisation of cardiac cells as a result of enhanced normal automaticity, abnormal automaticity or triggered electrical activity [1,2], or due to the formation of macro or micro re-entry circuits. Atrial fibrillation (AF) is a specific supraventricular arrhythmia in which a series of multiple microreentrant circuits form within the atria, resulting in a chaotic ventricular rhythm associated with the random selection of impulses that are conducted via the atrioventricular node to the ventricles [2,3]. Other supraventricular arrhythmias may be regular or irregular, depending on how the causal supraventricular impulses are conducted through the atrioventricular node.

Whilst there are many studies describing the natural history, treatment and prognostic significance of SVT and AF in people, our knowledge in cats is based solely on individual case reports [4–8] and a single retrospective study of 50 cats with AF [9]. In people [10] and large breed dogs [11,12], AF may occur in the absence of structural heart disease (known as ‘lone AF’). The latter is associated with reduced morbidity and mortality, compared to individuals with structural heart disease [11,13,14]. Lone AF appears to be rare in cats [5]. There is a positive association of AF with increases in atrial mass [15], and the majority of cats with AF have myocardial disease and severe left atrial enlargement. Most cats with AF are

male, consistent with the reported male predominance in feline cardiomyopathy [9,16].

The aim of this study was to describe the signalment, presenting complaints, cardiac phenotype and survival time in cats with SVT and AF. It was hypothesized that the prognosis of cats with AF would be worse than that of cats with SVT, and cats with AF would have a larger left atrium.

Animals, materials and methods

Retrospective study

Medical records from the Royal Veterinary College's Queen Mother Hospital for Animals' database were searched for cats examined between November 2004 and April 2014 using the key terms 'atrial fibrillation feline', 'AF feline', 'supraventricular tachycardia feline', and 'SVT feline'. Cats were included in the study if both an ECG recorded at 50mm/s at the date of diagnosis and an echocardiographic examination performed within 48 hours of the ECG recording were available for review. Cats without an ECG showing at least 2 leads were excluded. Information was collected on patient signalment, presenting signs, radiographic, ECG and echocardiographic findings, therapy and survival status.

All ECG traces from the date of examination were reviewed by a single board-certified cardiologist in order to confirm the presence of SVT or AF. For the purpose of this study, the average QRS depolarization rate over 3 seconds (the ventricular response rate) was referred to as heart rate (HR) and was measured from all ECGs. All of the cats had over 3 seconds of sustained SVT in the ECG recordings available for review for calculation of the average rate. To be classified as SVT, the rhythm had to demonstrate at least one of the following criteria

1. Criterion 1 for SVT: The presence of a sustained, narrow complex tachycardia lasting the entire duration of the stored ECG recording, with a regular R-R interval and HR greater than 260 bpm (Figure Ia supplementary data).
2. Criterion 2 for SVT: Demonstration of an abrupt onset or exit from a narrow complex tachycardia, either on the paper ECG recording or on subsequent telemetry (Figure Ib supplementary data).
3. Criterion 3 for SVT: Evidence of a persistent atrial depolarization at a rate greater than 260 bpm, with or without variable atrioventricular conduction ratio (Figure Ic supplementary data).
4. Criterion 4 for SVT: A wide complex tachycardia in which an intraventricular conduction disturbance was suspected, either due to the presence of a consistent atrioventricular relationship (i.e. a constant P-R interval could be identified) or in which QRS morphology was identical during sinus rhythm to the complex morphology documented during the tachycardia (Figure Id supplementary data).

For the purpose of this study, “SVT” refers to all types of supraventricular arrhythmias, excluding AF [1].

Atrial fibrillation was diagnosed when there was a clear absence of P waves in all recorded leads in association with a variable R-R interval or excluded in cats where P waves could be visualized [9].

The ECG traces were also reviewed for the presence of ventricular tachyarrhythmia. An ectopic complex was classified as ventricular in origin and premature if 1) the QRS complex was wide (>40ms) and bizarre with a large T wave with opposite polarity to the QRS complex, and 2) if they occurred prematurely when compared to the underlying rhythm (R-R’< R-R) [17]. Details of how wide complex tachycardia

with presumed intraventricular conduction disturbance were differentiated from true ventricular tachycardia have been outlined in the SVT rhythm criteria above.

All echocardiographic measurements were performed by a single board-certified cardiologist from stored two-dimensional images. All echocardiographic studies were acquired using the same ultrasound machine[a] and recorded by a board-certified cardiologist or supervised cardiology resident. Images were reviewed using a commercial analysis platform[b]. Each measured variable was calculated as an average of at least 3 and 5 different cardiac cycles for cats with SVT and AF respectively. A leading edge-to-leading edge technique was used to measure left ventricular (LV) wall thickness (septal and LV free wall) from the long and short-axis right parasternal 2-dimensional echocardiographic views at the level of the papillary muscles [18]. The LV wall measurements were obtained at end-diastole, defined as the first frame after mitral valve closure on the long-axis, or the frame at which left ventricular internal diameter (LVID) was greatest for short-axis views [19]. The maximal LV wall thickness was recorded as the highest value from averaged measures of the interventricular septum (IVS) and LV free wall measurements. Values ≥6mm were defined as left ventricular hypertrophy [18]. Two-dimensional measures of cardiac chamber internal dimensions were made using an inner edge-to-inner edge technique, at the boundary between the endocardial surface and blood pool [18]. End-diastolic LVID was measured from both long and short axis views. Left ventricular fractional shortening (LV FS%) was measured from M-Mode images obtained from a right parasternal short-axis view of the LV, obtained at the level of the papillary muscles. LV FS% was calculated using the following equation: LV FS% = [LVIDd–LVIDs] /LVIDd (where d=diastole and s=systole) [20]. The LVIDs was measured at the end of the T wave on the ECG [20]. Cats were considered to have

LV systolic dysfunction when LV FS% was ≤30% [19]. Assessment of right-sided cardiac dimensions was subjective; right atrial dilation was assessed by comparing right and left atrial areas from the right-parasternal long-axis view [20].

Left atrial (LA) size was assessed using two methods: LA diameter to aortic root diameter ratio (LA:Ao) taken from a two-dimensional image from the right parasternal short axis view, measured on the first frame after aortic valve closure and LA diameter (LAD), measured as the diameter of the left atrium parallel to the mitral annulus at the last frame before mitral valve opening, using a right parasternal 4 chamber long-axis view [21,22]. Left atrial enlargement (LAE) was present when LA:Ao >1.6 [18] and/or LAD ≥16mm. The presence of spontaneous echo contrast and/or an intra-cardiac thrombus was recorded. Each cat was assessed for the presence or absence of congenital cardiac disease, based on the opinion of the cardiologist reviewing the entire study.

Thoracic radiographs, when obtained, were evaluated by a board-certified radiologist at the time of presentation. Congestive heart failure status was determined by the presence of pulmonary infiltrates consistent with pulmonary edema on thoracic radiography and/or pleural effusion or pericardial effusion on echocardiography as determined by a board-certified cardiologist.

Survival data were obtained from clinical records, or where date or cause of death was not documented, referring veterinary practices were contacted between August and December 2014 to establish the outcome of each cat. Cardiac death was defined as animals that had been euthanized or died because of congestive heart

failure (CHF) or arterial thromboembolism (ATE) or died as a result of sudden cardiac death (SCD). Definitions used for these events were as follows: SCD was defined as being found dead without an obvious cause at home where the cat had been apparently well during the preceding 24 hours or as a witnessed event. Death due to CHF was defined as dying with dyspnea, crackles, cyanosis, fluid pouring out of the mouth and/or euthanasia due to becoming refractory to CHF medication. Death due to ATE was defined as death or euthanasia following a new episode of ATE or worsening of a current ATE episode [23].

Statistical analysis was performed using commercially available software[c]. Continuous data were assessed for normality using the Shapiro-Wilk test. Normally distributed data are presented as mean (± standard deviation) and non-normally distributed data are reported as median [range]. Differences between population characteristics of cats with SVT vs AF were compared using the two-sample t-test and Mann-Whitney U test for normally and non-normally distributed data respectively. Categorical variables were compared using Chi-squared test or Fischer's exact test as appropriate. A statistically significant result was defined as a p-value <0.05. Kaplan-Meier survival curves were generated to assess for impact of rhythm diagnosis, presence of ventricular arrhythmia, left atrial size (using LAD), HR and congestive heart failure status on cardiac death. Differences between groups were analysed using the Logrank (Mantel-Cox) test. Data was censored if death was due to unknown or non-cardiac reasons, or if they were still alive at the end of the study. Survival times are reported as medians (range).

Results

One-hundred-and-eight cats were identified from the clinical records database that had been diagnosed with SVT or AF. Fifty-four cats were excluded due to unavailability of a recorded ECG for review, while 10 cats were excluded due to corruption of the stored echocardiographic data, leaving 44 cats that were eligible for inclusion. There was no significant difference in sex (p=0.7), age (p=0.1), or breed (p=0.4) between cats with SVT or cats with AF. The majority (32/44) were male (see table 1). The breeds represented were domestic short hair (n=29), domestic long hair (n=5), Maine Coon (n=2), British Shorthair (n=2), Birman (n=2), Persian (n=1), Ragdoll (n=1), Devon Rex (n=1) and Sphynx (n=1).

Initial presenting signs were recorded for all 44 cats and are summarized in table 2. The most frequent presenting signs across both groups were respiratory distress and lethargy. All cats with AF had clinical signs. Only 2 cats were subclinical on presentation, both of which had SVT.

As expected, all cats with AF had a chaotic rhythm on auscultation, compared to 12 cats with SVT (p=0.01), likely due to the presence of sinus rhythm with numerous supraventricular ectopic complexes, numerous paroxysms of SVT or due to the presence of SVT with variable atrioventricular conduction at the time of auscultation. The group of cats with AF had a significantly slower HR documented on their ECG (220 beats per minute [180-260]) when compared to the group of cats with SVT (300 beats per minute [150-380], p<0.001).

Thoracic radiographs were available in 27 of the cats (13 cats with AF and 14 cats with SVT). Congestive heart failure was documented in 18 cats (11 cats with AF and

7 cats with SVT): 3 cats had pulmonary edema (1 cat with AF, 2 cats with SVT), 12 had pleural effusion (8 cats with AF, 4 cats with SVT) and 3 cats had both pleural effusion and edema (2 cats with AF and 1 with SVT), with no significant differences between groups (p=0.1). The presence of pericardial effusion was documented via ultrasound in 7 cats (1 with SVT and 6 with AF).

All cats with AF had echocardiographic evidence of left or right atrial enlargement, whereas 4 cats with SVT had no evidence of underlying cardiac disease. Of the latter, one cat (an 11-year-old MN DLH) was subsequently diagnosed with splenic haemangiosarcoma with metastases to both liver and omentum. Another cat (9-year-old MN DSH) had a history of chronic diarrhea and recent onset vomiting of unknown origin. Investigations revealed no significant biochemical or hematological abnormalities and urinalysis and abdominal ultrasound were unremarkable. No identifiable co-morbidities were found in the 2 remaining cats. The ECGs from these 4 cats are available for review as supplementary data online, (Figures II to V). When cats were classified according to severity of LAE using LAD, 7 cats had mild LAE (5 with SVT, 2 with AF), 15 cats had moderate LAE (7 with SVT and 8 with AF) and 12 cats had severe LAE (5 with SVT and 7 with AF). The following values were used for mild, moderate and severe LAE respectively; 16-18mm, 18-24mm and >24mm.[20] Only two cats had congenital heart disease, including a cat with SVT that had left ventricular hypertrophy, LAE and a ventricular septal defect (VSD) identified incidentally on echocardiographic examination and a cat with AF that had a double-chambered right ventricle, VSD and severe right atrial enlargement.

The proportion of cats with left ventricular hypertrophy was similar between groups which was documented in 14 cats with AF and 11 with SVT (p=0.2). Subjective right-sided eccentric +/- concentric right ventricular hypertrophy was more commonly identified in cats with AF (p=0.04), and of the 7 cats with right-sided cardiac changes (1 cat with SVT and 6 cats with AF), 6 had concurrent LAE. Left ventricular systolic dysfunction was identified in 8 cats, with equal numbers in both groups.

The majority of cats had LAE (see table 4), the proportion of which was similar between groups. Cats with AF had larger LAD (23.7mm [16.1-40.1] vs 19.1mm [12.8-31.4], p=0.02). There was no significant difference in LA:Ao between groups (p=0.08). The 4 cats with SVT and no LAE were the same cats detailed previously as having no evidence of structural cardiac disease. The only cat with AF and no LAE was the cat with a double-chambered right ventricle, VSD, and severe right atrial enlargement. Spontaneous echo-contrast was common, identified in 22% of cats (n=10). Six of these cats also had visible thrombi within the left atrial appendage.

Cardiac medications were administered to 43 of the 44 cats. The antiarrhythmic medications administered were diltiazem (n=15), atenolol (n=10) and sotalol (n=1) Antiarrhythmic medication was administered more commonly in cats with SVT than AF (16 cats vs 6 cats respectively, p=0.01). Antithrombotic medication (aspirin and/or clopidogrel) was administered in 23 cats and 29 cats were treated with furosemide.

Survival information was available for 40 cats (21 SVT and 19 AF), and the cause of death was known for 32 cats (15 SVT and 17 AF). The most common cause of death across both groups was refractory CHF, occurring in 47% of cats with AF and 43% of

cats with SVT (10 cats with AF and 10 cats with SVT). This was followed by ATE reported in 23% of cats with AF (n=5) and 13% of cats with SVT (n=3). Sudden cardiac death was reported in 2 cats (1 with AF and 1 with SVT). Median survival was 58 days [1 – 780] in cats with AF, and 259 days (2 – 2295] in cats with SVT (Figure 1, p=0.1). Cats with a lower HR did not survive longer until cardiac death than cats with a higher HR with either tachyarrhythmia, or when the population of cats was considered as a single group (Figure VI in supplementary data). Increased LA size (according to median LAD for cats with SVT, AF and all cats) did not predict worse survival (Figure VII in supplementary data). The presence of congestive heart failure at presentation was associated with a worse prognosis, (p=0.001, Figure 2) whilst the presence or absence of ventricular arrhythmia on resting ECG had no association with cardiac death (Figure VIII in supplementary data).

Discussion

The causal mechanisms, predisposing factors and natural course of SVT and AF are well described in people and to a lesser extent in dogs. Cats with SVT and AF have not been well-represented in the literature, and our study provides information on the characteristics and outcome in cats with these rhythm disturbances. Cats with AF are generally recognised as having a poor prognosis [9], so we have provided similar information in a contemporary cohort of cats with SVT as a comparison. This is the largest study to describe cats with SVT.

The signalment of this population was reflective of the high prevalence of cardiomyopathy, demonstrating that cats presenting with SVT or AF have a male predisposition and first present at a wide range of ages [9,16,24]. The most common

presenting sign across both groups was respiratory distress, most likely a sign of congestive heart failure, which is in agreement with previously published studies demonstrating that the primary presenting sign of cats with AF is decompensated cardiac disease [9].

The contribution and relevance of arrhythmia-induced remodeling to cardiac disease progression is poorly understood in cats. In dogs, SVT appears to be frequently associated with structural heart disease (65% of cases in one study) [25]. It is still not clear whether the structural changes seen in these cats are a consequence of pre-existing myocardial disease or due to the presence of chronic arrhythmias resulting in cardiac remodeling. Information regarding thyroid status and blood pressure were inconsistently recorded and therefore secondary cardiomyopathy could not be excluded in these cats. Hyperthyroidism is a known risk factor for the development of atrial fibrillation and supraventricular tachycardias in people (prevalence varies from 2-20%) [26]. In people, achievement of a euthyroid state is typically associated with restoration of sinus rhythm, especially in young patients and where duration of disease is not long [26]. Thyroid status was variably determined in our population of cats. Consequently, any association between hyperthyroidism and the presence of supraventricular arrhythmias could not be assessed and is considered a limitation of the study.

Left atrial diameter was used to assess LA size (in addition to LA:Ao) and was significantly larger in cats with AF than cats with SVT. A critical mass of atrial tissue is required to sustain the minimal number of circuits necessary to perpetuate AF [15], and consequently, this arrhythmia is frequently associated with conditions (e.g., hypertrophic, dilated or restrictive cardiomyopathy) that cause left or right atrial

dilation. This finding is also reported in the Côté *et al.* (2004) study that identified LAE in cats with concurrent AF [9]. Left atrial enlargement is associated with a poor prognosis in cats with acquired heart disease, and consequently, AF has been considered an end-stage event in cats with myocardial disease [27]. Left atrial size did not have an impact on overall survival in our study. This may be due to the small numbers of cats included in this population, as a larger study demonstrated a measurable effect of LA size on outcome [16]. There is one report of lone AF in a cat [5]. One cat with AF had normal LA size in our study, but in this cat, development of AF was attributed to severe right atrial enlargement (the cat had a double-chambered right ventricle and VSD).

The majority of cats with right-sided remodelling also had LAE. These changes could represent primary bilateral ventricular pathology (e.g. hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy affecting both ventricles), or might reflect remodelling consistent with a tachycardia-induced cardiomyopathy. Tachycardia-induced remodeling is a well-established pathological sequela to rapid-pacing in experimental models of cardiac failure in dogs and has also been documented secondary to naturally-acquired tachyarrhythmia in dogs [28]. Sustained tachycardia or paroxysms of any type of tachyarrhythmia affecting more than 15% of the daily heart beats may result in tachycardia-induced cardiomyopathy in people [29–31]. A single case report exists describing feline tachycardia-induced cardiomyopathy [5], in which follow-up longitudinal echocardiographic assessment showed progressive reduction in cardiac size in response to oral antiarrhythmic therapy. Unfortunately, few cats in this study had echocardiographic assessment prior to onset of the arrhythmia, longitudinal echocardiographic follow up and/or Holter ECGs to demonstrate whether or not rate control was adequate.

The data presented here show that SVT can be documented in cats without structural heart disease. In 2 of the 4 cats with no evidence of heart disease, concurrent systemic disease was also documented (metastatic splenic hemangiosarcoma, and chronic gastrointestinal disease of unknown origin respectively.) Gastrointestinal signs are frequently reported in dogs with SVT [25], however, it is not possible for us establish whether there is any link between the rhythm disturbances in these cats and their concurrent systemic signs. Two cats presented with paroxysmal supraventricular tachycardia and had no known co-morbidities, though further characterisation of the SVT could not be achieved. A potential mechanism for SVT in a young animal without structural heart disease is macroreentrant tachycardia involving an accessory pathway, but the electrocardiographic features for this condition have not been well described in cats. Ventricular pre-excitation was not identified in any cat in this study and the current practical limitations in performing diagnostic electrophysiological studies makes it challenging to achieve a definitive diagnosis in cats with SVT [3].

The median HR obtained from the ECG recordings was found to be higher in cats with SVT than AF (300 bpm and 220 bpm respectively), which is perhaps unsurprising given the diagnostic criteria for this study for cats with SVT (i.e. the presence of an sustained narrow complex tachycardia, with a regular R-R interval and HR greater than 260 bpm). The median HR of 220 bpm in cats with AF was similar to the rate that was published from a larger group of cats with this arrhythmia [9]. It is interesting to note that medications aimed at reducing HR were uncommonly prescribed to cats with AF in this population despite the fact that control of average HR is considered to be an important therapeutic target in both people and dogs. It is also impossible for us to conclude whether the administration of medication to these

323 'treated' cats had an influence on survival due to inconsistencies in treatment
324 recording in relation to ECG traces in the hospital and lack of subsequent re-
325 examinations following the introduction of treatment. Recently published
326 retrospective data in dogs has suggested that a lower average HR in dogs with AF is
327 associated with improved survival [32] and it is not known whether the same would
328 be true in cats. This study did not document a statistically significant association
329 between HR and survival in cats with AF or SVT, or when the population was
330 considered as a whole, although it is possible that this study was under-powered to
331 achieve statistical significance. Furthermore, the impact of medication on survival
332 remains unknown due to incomplete information available from the clinical records;
333 specifically, regarding the timing of medication administration in relation to
334 acquisition of the recorded ECG traces, doses of medications, lack of 24-hour Holter
335 ECG analysis and infrequency of re-examination following the introduction of
336 treatment.
337
338 In this group of cats, the documentation of transient ventricular arrhythmia on 6-lead
339 paper trace ECGs was not associated with worse survival. In recent studies
340 evaluating clinical risk markers for HCM in people, non-sustained ventricular
341 tachycardia proved to be a significant independent risk factor for SCD, especially in
342 the young [33]. We know from previous studies that cats with myocardial disease
343 have more frequent and complex ventricular arrhythmia than normal cats [34],
344 however further research, ideally with Holter ECG data and a larger number of cats,
345 would be required to determine whether these arrhythmias are associated with
346 increased risk of SCD or whether they influence long-term survival.

There were many limitations of this study, some of which relate to the inherent challenges associated with the recording and interpretation of feline ECGs, including low amplitude voltages, and motion/purring artifacts. At a HR > 260 bpm, short R-R intervals can make it challenging to differentiate truly irregular rhythms from regular rhythms [9], and some of the ECGs may have been incorrectly classified. In particular, small p' (supraventricular depolarizations not arising from the sinoatrial node) wave amplitudes made further classification of the SVT impossible in many cases and so was not attempted in this current study. In some cases, irregular SVT (e.g. due to multifocal atrial tachycardias or atrial flutter) may have been misclassified as AF due to an inability to identify p' or flutter waves. Furthermore, criteria to define SVT were extrapolated from those used in dogs due to lack of well-established criteria in cats. An arbitrary rate of 260 bpm was used to define SVT; however, it is possible that some of these cats may have had a physiological sinus tachycardia. Conversely, some cats with a true SVT but a HR less than 260 bpm may have been mistakenly excluded. Comparing cats with SVT to those with AF carries with it a number of inherent limitations. Atrial fibrillation was presumed to be sustained in all cats presenting with this arrhythmia. Holter ECGs were not performed in any cat and therefore the frequency and duration of SVT is unknown. Consequently, it is challenging for us to draw conclusions regarding the impact of this arrhythmia on myocardial remodeling and possible CHF, for example, and comparing survival data without Holter data therefore may be inappropriate. Identification of ventricular arrhythmia from a 6-lead paper trace ECG is inferior to Holter ECG assessment, and therefore underreporting of ventricular arrhythmia is likely to be present in this group of cats. Further studies regarding the prognostic significance of HR in the hospital and home environment of cats with SVT and AF

are warranted as this may provide additional information with regards to optimizing therapy. Thoracic radiographs were not performed in all cats and therefore some cats may have been misclassified regarding their CHF status.

Almost half of the cats diagnosed with SVT or AF according to the clinical records system had to be excluded due to missing ECG records from the patient file. A further 10 cats were removed as the storage discs containing the echocardiographic images had become corrupted. This could affect the application of findings from this study to a wider population of cats and importantly the small group sizes may have limited the statistical power of the analyses. Further studies involving a larger number of cats are therefore warranted.

Conclusions

Supraventricular tachycardia was as common as AF in our hospital population, despite the paucity of case reports in the literature. All cats with AF had underlying structural heart disease, whereas some cats with SVT had normal cardiac chamber dimensions.

Conflicts of interest statement:

The authors do not have any conflicts of interest to disclose.

Footnotes:

a) Vivid 7, General Electric Medical Systems Ultrasound, 71 Great North Road, Hatfield, AL9 5EN, United Kingdom.

b) Echopac, General Electric Medical Systems Ultrasound, 71 Great North Road, Hatfield, AL9 5EN, United Kingdom.

c) BM SPSS Statistics 21.0 for Windows 7, IBM (UK) Ltd, Portsmouth, UK; GraphPad Prism 6, GraphPad Software Inc, San Diego, CA

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Figure 1.

Kaplan-Meier curves to explore the difference in median survival time between cats with supraventricular tachycardia (SVT) and cats with atrial fibrillation (AF)

Figure 2.

Kaplan-Meier curves to explore the difference in median survival time between cats with and without signs of congestive heart failure (CHF)

Demographic findings			
Variable	**AF (n=21)**	**SVT (n=23)**	**p-value**
Male	16	16	0.7
Pedigree	4	6	0.7
Median age (months)	121 (40-174)	84 (6-196)	0.1
Median HR (bpm) established from paper trace ECG	220 (180-260)	300 (150-380)	<0.001

Table 1. Demographic data for cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to presenting rhythm diagnosis. HR: Heart rate

Presenting complaint	AF (n=21)	SVT (n=23)	p-value
No clinical signs	0 (0.0%)	2 (4.5%)	0.5
Respiratory distress	6 (13.6%)	4 (9.1%)	0.5
Lethargy	6 (13.6%)	3 (6.8%)	0.3
Collapse	2 (4.5%)	6 (13.6%)	0.2
Hindlimb paresis	3 (6.8 %)	2 (4.5%)	0.7
Ascites	2 (4.5%)	1 (2.3%)	0.6
Weight loss	1 (2.3%)	2 (4.5%)	>0.9
Cough	1 (2.3%)	0 (0.0%)	0.5
Inappetence	0 (0.0%)	1 (2.3%)	>0.9
Vomiting	0 (0.0%)	1 (2.3%)	>0.9
Weakness	0 (0.0%)	1 (2.3%)	>0.9

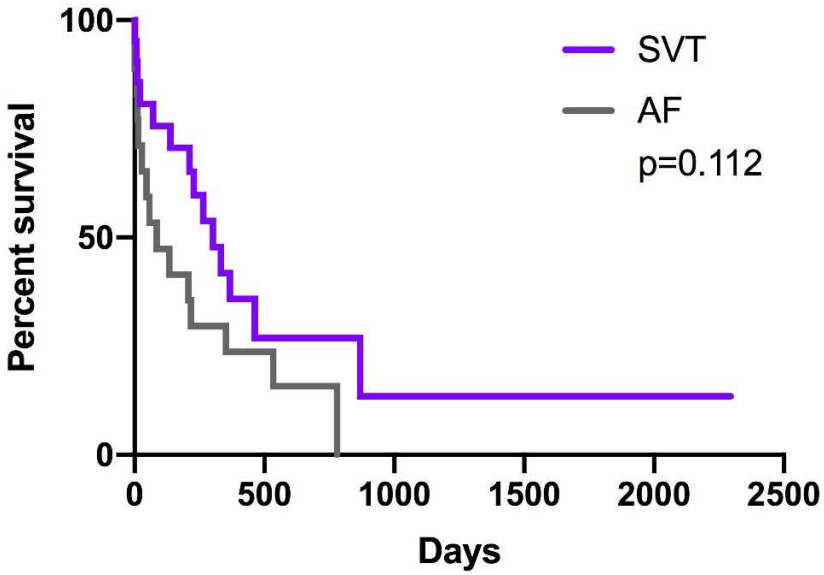
Table 2. Presenting signs of cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to presenting rhythm diagnosis.

Echocardiographic findings			
Variable	**AF (n=21)**	**SVT (n=23)**	**p-value**
Normal heart	0	4	0.1
Congenital disease	1	1	>0.9
LVH	14	11	0.2
LAE	20	19	0.1
SEC/thrombus	5	5	>0.9
Right heart disease	6	1	0.04
Systolic dysfunction	4	4	>0.9

Table 3. Cats with supraventricular tachycardia (SVT) and atrial fibrillation (AF) grouped according to phenotypic findings on echocardiography. LAE: left atrial enlargement (LA:Ao >1.6 and/or LAD>16mm); LVH: left ventricular hypertrophy; SEC: spontaneous echo contrast

Echocardiographic findings			
Variable	**AF**	**SVT**	**p-value**
LVPWDd LAX (mm)	7.1 (3.6-10.5)	5.35 (3.9-12.1)	0.2
IVSd SAX (mm)	5.55 (3.1-8.4)	5 (3.7-8.5)	0.1
IVSd LAX (mm)	6 (3.9-9.0)	5.3 (4.1-9.9)	0.6
LVPWd SAX (mm)	6.16 +- 0.49	6.1 +- 0.40	0.9
LVID LAX (mm)	15.79 +- 0.72	14.98 +- 0.87	0.5
LA/Ao ratio	2.63 +- 0.16	2.22 +- 0.15	0.08
LAD (mm)	**23.7 (16.1-40.1)**	**19.1 (12.8-31.4)**	**0.02**
FS %	37.29 +- 4.92	38.88 +- 3.88	0.8

Table 4. Specific echocardiographic measures of left ventricular wall thickness, left atrial size and systolic function. FS%: fractional shortening; IVSd SAX: Interventricular septum in diastole, short-axis; LA/Ao: left atrial to aortic ratio; LVID LAX: left ventricular internal diameter, long-axis; LVPWDd LAX: left ventricular posterior wall diameter in diastole, long-axis; LVPWd SAX: left ventricular posterior wall diameter in diastole, short-axis.


100
50
0
Percent survival
0
500
1000
1500
2000
2500
Days
SVT
AF
p=0.112