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JOURNAL TITLE: Journal of Feline Medicine and Surgery

PUBLICATION DATE: August 2018

PUBLISHER: SAGE Publications

DOI: 10.1177/1098612X17723775

Title: **Magnetic resonance imaging and clinical characteristics of suspected cerebrovascular accident in 9 cats**

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Keywords: Feline, Brain, Infarct, Encephalopathy, Ischaemia, MRI, Cerebrovascular accident.

The authors received no financial support for the research, authorship, and/or publication of this article.' The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Abstract

Objectives: Cerebrovascular accidents (CVAs) are infrequently reported in cats. To date, clinical characteristics, including lesion localization and magnetic resonance imaging (MRI) findings have only been reported in two cats. The aim of the current study is to document MRI findings in cats presenting with CVAs over an eleven-year period. Cases were reviewed according to initial clinical presentation, subsequent physical and neurological findings, predisposing systemic disease and short and long (when available) term outcome with a view to identifying any typical pattern in disease occurrence.

Methods: Patient records of cats presenting to a single referral center from January 2005 to September 2016 with acute onset, non-progressive (after 24 hours) intracranial signs compatible with CVA and where an MRI was performed within 72 hours were retrospectively reviewed.

Results: Nine cats met the inclusion criteria. All cats had ischaemic CVAs (presumptively diagnosed in eight cats and confirmed in one cat following post mortem examination). No cases of haemorragic CVAs were identified. Four cats presented with territorial infarcts that were confined to the territory of the rostral or caudal cerebellar arteries (n=4). Lacunar infarcts were identified in five cats in the location of the cerebrum (n=1), the thalamus/midbrain (n=2) and the medulla oblongata (n=2). Concurrent systemic disease was identified in most (8/9). In the present study short-term prognosis was favourable and 8/9 cats survived to 48 hours following admission.

Conclusions and relevance: CVAs in cats occur in the same vascular territories as in dogs and have similar MRI features. This study notes that the presenting cats had a high likelihood of concurrent disease (8/9 cases) but had a favorable short-term prognosis, if neither the clinical presentation nor concurrent disease were severe.

Introduction

Cerebrovascular accidents (CVAs) are defined as a disruption in cerebral blood supply leading to neurological dysfunction and are classically associated with clinical signs that are peracute in onset and non-progressive after 24 hours (1). CVAs, a temporary or permanent reduction in cerebral blood flow, are the most common cause of cerebrovascular disease in both the human population and in veterinary species, and occur either secondary to a local thrombus or a thromboembolism in the case of ischaemic CVAs or to intracranial blood vessel rupture in haemorrhagic CVAs (1-4). Whilst cerebrovascular disease is the subject of multiple reviews in canine patients (5-8), it has rarely been reported in the cat (9, 10). In fact, signalment, concurrent systemic disease and outcome of ischaemic and haemorrhagic CVAs have all been subject to review in dogs (5, 6, 11, 12). To date, clinical characteristics, including MRI findings of ischaemic CVAs, have been reported in just two cats ante mortem (9). Cats with cerebrovascular disease have also been described in relation to histopathological features, including seven cases of ischaemic CVAs (six diagnosed following post mortem examination and a further one by computed tomography guided biopsy) and five haemorrhagic CVAs (10). Post mortem reports introduce inherent bias in selecting the most severely affected cases. Therefore it is unclear whether these previous reports represent an accurate reflection of CVA distribution and frequency in cats.

The MRI characteristics of CVAs in dogs are well described (5) whereas in cats they are limited to a single report of two cases of rostral cerebellar infarction (9). Species-specific adaptations in cerebral blood supply are well described, for example, it is known that the major blood vessels supplying the arterial circle in the canine and feline populations differ

(3, 10, 13-15). Compared to cats, the vertebral arteries supply a much greater proportion of the canine brain, including the thalamus and caudal-most aspect of the cerebral cortex (15-17). In cats the vertebral artery irrigates only the caudal-most part of the medulla oblongata (14). Whether these anatomical differences influence the frequency or distribution of infarcts in cats compared to dogs or alter typical MRI features is currently unclear.

The aim of the current study is to report the MRI features, clinical characteristics, (inclusive of concurrent systemic disease) and short and long-term outcome, of CVAs in the feline population presenting to a single referral hospital over an eleven-year period.

Materials and Methods

Approval for the study was obtained from the Clinical Research Ethical Review Board at the Royal Veterinary College. Medical records of all cats presenting to a single referral hospital (Queen Mother Hospital for Animals, Royal Veterinary College) between January 2005 and September 2016 with acute onset, non-progressive (after 24 hours) intracranial signs were retrospectively reviewed for inclusion in the study. From those, only cats that presented within 48 hours of onset of clinical signs, had full physical and neurological examinations and underwent MRI within 72 hours of presentation were included. Clinical records were systematically reviewed to take into account the following factors: (i) presentation, (ii) clinical progression and (iii) additional diagnostics tests used to assess for concurrent disease. Where available, cerebrospinal fluid (CSF) analysis (cell count, cytological examination, total protein concentration determination and infectious disease testing) was recorded. Of those with CSF analysis available, only those that had results consistent with

previous reports for CVA, specifically normal CSF analysis or mild neutrophilic or mononuclear pleocytosis (<30cells/µl) (6) were included.

All MRI sequences were obtained with a 1.5 Tesla unit (Intera Pulsar System, Philips Medical Systems, Reigate, UK). A complete MRI series included the following sequences: T2-weighted images (T2W) in the sagittal and transverse planes and pre and post contrast T1-weighted images (T1W) in transverse planes. Other sequences were analysed when available and included T2W and T1W sequences in the dorsal plane, fluid- attenuated inversion recovery (FLAIR), GRE and diffusion weighted imaging (DWI). Cats that received corticosteroids or antibiotics, other than those that received a single dose, were excluded from the study. The post-contrast enhancement was categorised as none, mild when contrast enhancement was hypointense compared to the fat tissue on T1W images and marked when the contrast enhancement was isointense to the fat tissue on T1W images. The contrast enhancement was also categorised as homogeneous when there was uniform enhancement and heterogeneous when it was dissimilar throughout the enhanced area. The degree of contrast enhancement in lesions was assessed and classified as none, mild or marked and heterogeneous or homogeneous by two independent reviewers.

In all cases, a board certified veterinary radiologist (RD) and a board certified veterinary neurologist (EB) independently reviewed the MRI sequences available. A diagnosis of CVA was made when both reviewers independently agreed on a diagnosis, according to imaging criteria outlined previously (5). In the case of ischaemic CVAs, this included lesions that were predominantly confined to the grey matter and limited to the vascular territory of a main

artery (territorial CVA) or perforating arteries (lacunar CVA). Lesions were predominantly homogeneous in appearance and hyperintense on T2W and FLAIR sequences with well-demarcated boundaries to adjacent brain parenchyma. Minimal or no mass effect was noted. Cats presenting with suspected CVAs affecting thalamic arterial blood supply were further categorised in accordance with a classification scheme previously applied in dogs (4) that subdivides thalamic infarcts by location and vascular territory into (i) paramedian, (ii) dorsal extensive, and (iii) ventrolateral infarcts. In the case of haemorrhagic CVAs, MRI sequences were reviewed to identify the presence of acute or early subacute intraparenchymal haemorrhage identified by iso- to hyperintense signal on T2W images, and iso- to slightly hyperintense signal on T1W images. The presence or absence of mass effect was recorded. GRE sequences were reviewed when available and considered compatible with haemorrhage when lesions were hypointense (21). Cats with other brain pathology were excluded.

Reviewers (EB, RD) further defined infarcts according to: location (cerebrum, thalamus, midbrain, cerebellum (divided into rostral, and caudal cerebellum according to their location in relation to the primary fissure), pons, medulla oblongata) and arterial territory in the cases of ischaemic CVAs classified as territorial (occlusion of a main artery, including, rostral cerebral, middle cerebral, caudal cerebral, rostral cerebellar, caudal cerebellar, or vertebral) or lacunar (occlusion of a small perforating arterial vessel, including, superficial and deep perforating arteries)).

Short-term outcome was defined as survival or non-survival at 48 hours after presumptive diagnosis of a CVA. Long-term outcome was assessed, when possible, at a minimum of 4

weeks after diagnosis. Follow-up information was obtained by telephone consultation with the owner. Long–term outcome was defined as dead or euthanized, poor (severe neurological deficits or no improvement in neurological signs since presentation), good (clinical signs improved since discharge but neurological deficits are still present) or excellent (clinical signs resolved since discharge and the cat considered normal).

Results

Fourteen cats presented to the Queen Mother Hospital for Animals, between January 2005 and September 2016 with suspected CVA. Two cats were excluded since their clinical signs improved and they were discharged without MRI. A further three cats were excluded, two due to the additional finding of a meningioma and a further was excluded having received a prolonged course of corticosteroids on the basis that inflammatory, infectious and neoplastic disorders could not be excluded in this case. A total of nine catswere evaluated for this study. Haemorrhagic CVAs were not identified in any case. In the remainder of the text the term “CVA” in this study should be construed as referring only to ischaemic CVAs (presumptively diagnosed in eight cats and confirmed in one following post mortem examination). All eight cases with suspected CVAs displayed improvements in clinical signs prior to discharge. In the confirmed case, the owner elected for euthanasia owing to the severity of clinical signs following cardiorespiratory arrest despite successful resuscitation. In this case a cerebellar ischaemic CVA was confirmed post mortem.

Of those cats included there were four (44.45%) neutered females, one (11.10%) sexually intact female and four (44.45%) neutered males with a median age of presentation of 12

years (range, 2 years, 2 months-16years). Breeds represented included Domestic Short Haired Cat (n=7), Domestic Long Haired Cat (1) and Bengal Cat (1).

Imaging findings

All cats presented within 48 hours and were imaged within 72 hours of onset of clinical signs. T2W sequences in the sagittal and transverse planes and pre and post contrast T1W images in the transverse plane were available in all cats. Additional sequences were also obtained, including T2W dorsal (n=2), FLAIR (n=6), GRE (n=8), and DWI (n=4). All lesions were focal and well to marginally well demarcated to adjacent brain parenchyma. Lesions were present in the location of a vascular territory and predominantly restricted to grey matter in all but one case. In that case the cerebral lesion extended from the head of the caudate nucleus to the white matter of the internal capsule (Figure 1A,C, Figure 2B,C). In all cases, lesions were hyperintense to normal grey matter on T2W and, when available, on FLAIR sequences and were either slightly heterogeneous in the periphery (n=1) or homogeneous (n=8) in appearance. In T1W sequences lesions were isointense (n=3), hypointense (n=4) or hypo-isointense (n=2) to normal grey matter. Lesions either exhibited no contrast enhancement (n=3), mild to no contrast enhancement (n=4) or mild enhancement that was restricted to the periphery of the lesion (n=2). Lesions were hyperintense to normal grey matter on GRE (n=5) with no signal void. Where DWI was available (n=4), lesions were hyperintense to normal grey matter on DWI and hypointense on the apparent diffusion coefficient (ADC) map, indicative of restricted diffusion, consistent with cytotoxic oedema (22) (Figure 3). DWI was non-diagnostic in one case. There was no (n=5) or minimal (n=4) mass effect in all cases.

The distribution of suspected CVA was determined based on MRI findings and as follows: cerebral (extending into the thalamus, (1/9)), thalamic/midbrain (2/9), cerebellar (4/9), and those restricted to the medulla oblongata (2/9) (Table 1)]. Lesions were restricted to the following vascular territories as determined by MRI: lenticulostriate arteries of the middle cerebral artery (1/9), perforating arteries of the caudal cerebral artery (2/9), rostral cerebellar artery (2/9), caudal cerebellar artery (1/9), rostral and caudal cerebellar arteries (1/9) and basilar artery (2/9) (Figure 1).

The cerebral lesion was identified at the level of the head of the caudate nucleus and extended into the adjacent internal capsule (Figure 2B,C) in the arterial territory of the lenticulostriate arteries (a branch of the middle cerebral artery) (Figure 1A,C). The thalamic and midbrain lesions were identified in the right ventral thalamus at the level of the pituitary gland and in the left midbrain at the level of the rostral colliculi (Figure 1B, Figure 2D, E, F, G). These presumptive CVAs were located in the territory of perforating arteries (branches of the caudal cerebral artery) namely the proximal and caudal perforating arteries, respectively (Figure 1A, B). Those confined to the proximal and caudal perforating arteries affecting the thalamic arterial blood supply were further defined topographically as paramedian, according to the classification scheme applied to dogs (7).

Lesions were identified in the cerebellum of four cats (Figure 1B). In two cats a lesion was identified in the territories of the right and left rostral cerebellar arteries affecting the rostral and middle cerebellum (Figure 2H,I) whilst another cat had a lesion in the right caudal cerebellum in the territory of the right caudal cerebellar artery (Figure 2J, K). One cat

had a lesion in the vascular territory of both the rostral and caudal cerebellar arteries (Figure 1A,B). A further two cats had CVAs in the medulla oblongata in the territory of the perforating branch of the basilar artery (Figure 1A,B, Figure 2 L, M). One cat had a lesion in a rostrolateral position in the upper rostral medulla oblongata whilst a further cat had a lesion in the dorsomedial location in the middle medulla oblongata.

Clinical signs

In all cases, clinical signs reflected neurological dysfunction and varied dependent on the location of the CVA (summarized in Table 1). Specifically the cat presenting with a presumptive diagnosis of CVA in the middle cerebral artery and those cats presenting with a presumed CVA in the caudal cerebral artery exhibited signs of forebrain dysfunction, those located in the rostral and caudal cerebellar arteries exhibited signs of cerebellar dysfunction and those with a presumed CVA located in the basilar artery exhibited signs of brainstem dysfunction.

General physical examination was unremarkable in all but three. Two cats had a grade II/VI heart murmur and one had a gallop rhythm noted on thoracic auscultation. Those two cats with a heart murmur were diagnosed with systemic hypertension with bilateral retinal haemorrhage noted on fundoscopy. Overall, concurrent disease was identified in 8/9 (89%) cats (Table 2). Of these, four cats (44%) had hypertrophic cardiomyopathy (HCM). Of those with HCM two had normal left atrial size, one had a dilated left atrium with severe left ventricular hypertrophy and ventricular outflow tract obstruction, the other had marked dilatation of the left atrium with poor left atrial formation and a thrombus was also identified. One of the two cats with HCM and normal left atrial size had concurrent systemic hypertension as outlined above. Of the five cats without a diagnosis of HCM, two had HCM

ruled out by echocardiogram although left ventricular hypertrophy was noted on post mortem examination in one. Two did not undergo diagnostic cardiac ultrasound or post mortem examination (HCM diagnoses therefore are unknown, one cat in this group had a grade II/VI heart murmur as mentioned previously). Of those cats that were not diagnosed with HCM, two had pulmonary masses. One cat was diagnosed with a mass in the right caudal lung lobe that was suspected to have metastasised to a digit. Subsequent cytological examination of the suspected metastatic mass was consistent with a carcinoma suspected to have metastasised from a primary pulmonary carcinoma. The cat was euthanised due to a poor prognosis, however, post mortem was not performed. A pulmonary adenocarcinoma was also identified in one cat following post mortem examination (euthanised following cardiopulmonary arrest after a rostral cerebellar CVA). There were no reported clinical signs attributable to the mass on presentation. One cat had been diagnosed with hyperthyroidism one year prior to presentation to the referral hospital and was on carbimazole. The thyroid status of the cat at the time of presentation was unknown. In that case, bilateral otitis media was also detected by MRI.

Cerebrospinal fluid (CSF) findings

CSF was available in 4/9 of cases. All cats had results consistent with previous reports for CVA and were therefore eligible for inclusion into the study. Of these CSF was reported as normal in 3/4 (Total nucleated cell count (TNCC) <5mm^3, protein <0.25g/l). An increased TNCC count was identified in one cat (TNCC 18mm^3, RBC 0mm3, protein 0.01g/l). In that case *Toxoplasma gondii* was negative by polymerase chain reaction in CSF. A differential count in this case revealed 3 nucleated cells, specifically 2 large mononuclear cells and one macrophage with no sign of an aetiological agent.

Outcome

Most cats (8/9, 89%) had a good short-term prognosis (survived to 48 hours). One cat with a suspected rostral cerebellar CVA was euthanised and the presumptive diagnosis of a cerebellar CVA according to MRI findings was subsequently confirmed by post mortem examination. The cat had initially suffered a number of cardiorespiratory arrests following anesthesia and, although each time was successfully resuscitated, the cat was subsequently euthanised. Post mortem examination in this case revealed moderate left ventricular hypertrophy with evidence of renal infarcts and a pulmonary adenocarcinoma with angioinvasion. One cat survived to 48hrs, and was discharged following an improvement in clinical signs, however was re-admitted five days after initial discharge due to inappetence. Further investigation revealed a suspected primary pulmonary mass with likely secondary metastasis to a digit. Cytological analysis of the metastatic mass was consistent with a carcinoma with evidence of necrosis and osteolysis. This cat was euthanized due to poor prognosis. Long term follow up information was available for 2/7 (29%) remaining cases ranging from 5- 10 weeks. One cat was deemed to have an excellent outcome following discharge according to owners (complete resolution of clinical signs) and one cat had a good long-term outcome since there was a reported improvement in clinical signs, however some neurological deficits were still present (mild head tilt and mild vestibular ataxia).

Discussion

This study provides the most comprehensive overview to date of MRI and clinical characteristics of CVAs in cats. It is the first ante-mortem study that reviews MRI characteristics of naturally occurring CVAs in the feline population other than CVAs

occurring in the cerebellum. In agreement with a previous post mortem study (10), these data demonstrate that feline CVAs can occur in a number of vascular territories.

The retrospective nature of the study introduces significant limitations. In a recent study on dogs with histologically confirmed intracranial disease, MRI was 97.7% specific for identification of cerebrovascular lesions (23), however, interobserver agreement in CVAs was only fair and as such, strict inclusion criteria were applied to this dataset i.e. improvement of clinical signs in the absence of specific treatment in all cases where post mortem examination was unavailable. In the present study a full MRI sequence was available in all cases (T2W in the sagittal and transverse planes and pre and post contrast T1W in transverse planes). Although additional MRI sequences provide further support of a diagnosis of CVA, such as DWI, these sequences were only obtained in four of nine cats in this study. In prospective studies the inclusion of additional MRI sequences such as perfusion-weighted imaging (PWI) - a sequence that compliments DWI by permitting analysis of the blood supply of the tissue (24) - may also be useful given that post mortem confirmation is rarely available.

It is understood that the incidence of CVAs in cats and dogs is significantly lower when compared with the high incidence of strokes in the human population (approximately 795,000 individuals affected in the USA per year (25)). It has previously been proposed that the lower incidence of CVAs in dogs compared to humans is, in part, related to differences in cerebral blood supply. Specifically dogs have a higher number of intra-extracranial arterial anastomoses (16), which reflects an anatomical variation that is also recognised in cats (26). Thus species-specific adaptations of the cerebral blood supply present a credible

explanation for differences in prevalence and severity of CVA. Here, nine cats with suspected CVA were identified in a single referral center over an eleven-year period (comparative to 40 cases in dogs in a single report over a 52 month period (5)) Given the lack of data on the total population of cats and dogs presenting to the respective referral centres, it is unclear if this comparison infers a higher incidence of CVAs in cats compared to dogs. However, assuming the data represents some evidence of differentiation between the species with respect to CVA occurrence, literature has to date failed to offer an explanation. As stated, in common with dogs, cats have a high number of intra-extracranial anastomoses (13, 14), but despite this commonality, the cerebral blood supply differs between the dog and the cat. Specifically, the main arterial blood supply to the brain differs; the internal carotid and basilar arteries are the major contributors to the cerebral arterial circle in dogs (15), whilst the maxillary artery (derived from the external carotid artery) supplies all but the caudal region of the medulla oblongata (to the caudal boundary of the trapezoid body) in cats (13, 14, 26). It remains unclear whether this difference protects the feline population against CVAs, and if so how, or whether the lower incidence can be attributed to a different reason, for example, prevalence of predisposing disease. It is possible that fewer cats are referred for further investigation of CVAs; either due to less severe and rapidly improving or resolving neurological signs or due to significant concurrent medical conditions resulting in owners more readily electing euthanasia. Further surveys of first opinion (primary) practices are required to investigate this possibility.

Whilst supratentorial territorial CVAs are most commonly reported in human patients, with cerebellar CVAs accounting for approximately 1.5% of all strokes only (27), by contrast and in common with dogs, this study suggests that territorial CVAs in cats are more likely to

occur in the cerebellar arteries (5). However, caution should be taken in overstating this comparison due to the small sample size in this study. Four cats presented with territorial CVAs, all of which occurred in the cerebellum, whilst five cats had lacunar CVAs. A previous ante mortem study also identified rostral cerebellar CVAs in two cats (9). Frequency of territorial CVAs in the aforementioned ante mortem report and in this study are in contrast to an earlier post-mortem study, which did not identify a single CVA in the cerebellum (10). That study reviewed seven cats with ischaemic CVAs and instead identified five instances of territorial infarcts in the region of the cerebrum and brainstem in addition to two lacunar infarcts affecting the cerebrum and brainstem (10). The contrast between the current report and previous post mortem studies may be due to inherent differences in selection of cases in ante mortem versus post mortem studies with those included in the latter more likely to have severe neurological signs or unconnected and potentially severe underlying disease and/or clinical signs. Cerebellar infarcts appear to predominantly occur in the territory of the rostral cerebellar artery in dogs. To the author's knowledge there has only been a single case report describing a suspected caudal cerebellar artery infarct in a dog (28). The current report identifies an equal incidence of infarcts in both the rostral and caudal cerebellar territories in cats (n=3 in each case). This difference in distribution cannot be ascribed to a differentiation in the direct blood supply of the cerebellum between the species, given the blood supply of the cerebellum in cats is similar to dogs; the rostral and caudal cerebellar arteries supply the rostral and caudal cerebellum, respectively. However, differences have been noted in the blood supply of the caudal brainstem from which the caudal cerebellar artery is derived (26). In common with dogs, cats have extensive anastomoses supplying the brainstem derived from the cervical region (26). In the dog (but not in the cat) the occipital artery provides significant blood supply to the brainstem by contributing to the vertebral

artery (26). By contrast, in cats the basilar artery arises in the caudal medulla derived solely from the vertebral arteries without contribution from the occipital artery. It is plausible, although not proven dispositive, that this anatomical divergence in brainstem blood supply contributes to the potentially more frequent instances of caudal cerebellar CVAs in cats given the degree of collateral blood supply in the dog.

Cerebellar infarcts in human patients are, in general, most commonly linked to cardiac disease. Cerebral embolism is reported as the prevailing cause of an infarct in the superior cerebellar artery (equivalent to rostral cerebellar artery) (27), which is predominantly linked to cardiogenic causes such as valvular disease, dysrhythmias, and myocardial infarction (29) whilst posterior inferior cerebellar infarcts (equivalent to the caudal cerebellar artery in cats) were caused by cardiogenic embolism and posterior circulation arterial disease, including atherosclerotic disease (30) (secondary to occlusion or stenosis of the vertebral or the posterior inferior arteries) in equal proportions (27, 31). In this study, HCM was identified in four cats with concurrent systemic hypertension reported in one. Left atrial enlargement, a sequel of left ventricular hypertrophy in HCM, leads to secondary blood stasis and as such, predisposes cats to thromboembolic disease. Of the four cats diagnosed with HCM included in the present study, left atrial enlargement was identified in two and of these, a thrombus was identified in one. That cat, presumptively diagnosed with a CVA in the caudal cerebellar artery, had marked left atrial dilation with poor left atrial function. In the absence of left atrial enlargement, left ventricular hypertrophy is unlikely to lead to thromboembolic disease and therefore HCM is likely an incidental finding in the two remaining cases. One cat with caudal and rostral cerebellar CVAs had mild HCM with no dilation of the left atrium.. Of the remaining two cats with presumed cerebellar CVAs, one

had a primary pulmonary mass and the other had been diagnosed with hyperthyroidism one year earlier and was receiving carbimazole. The thyroid status of this cat was unknown and a full echocardiogram could not be performed, although a limited examination revealed mild septal hypertrophy deemed unlikely to be clinically significant. It remains unclear whether correlation noted in this study between concurrent disease and CVAs is causative and/or a factor that influences the site of infarction in cats or merely coincidental given the median age of the cat population is 12 years.

Two cats presented with presumptive CVAs in the medulla oblongata, which is a rare finding in dogs (5). In fact, it has only been reported in three dogs with multifocal lesions with presumptive CVAs occurring in both the thalamus and in the medulla oblongata (5). This is in common with human patients where CVAs in the medulla oblongata are rare (19). The occurrence in cats appears higher, accounting for 2/9 of cases in this study. The blood supply of the caudal brainstem differs between dogs and cats with the vertebral artery receiving a contribution from the occipital arteryin dogs. The degree of collateral blood supply therefore offers a plausible explanation for the apparent low prevalence of medullary CVAs in the dog.

Suspected thalamic CVAs were identified in two cats secondary to a lacunar CVA affecting the proximal perforating artery. These suspected CVAs were further classified as paramedian, the most common thalamic CVAs noted in dogs (7). In dogs with presumed thalamic CVAs, the most common presenting signs were attributable to vestibular dysfunction; vestibular ataxia was reported most frequently irrespective of the position of

the thalamic CVA. Vestibular ataxia was not noted in either cat although one, with a lesion in the territory of the caudal perforating artery (midbrain), was non-ambulatory and displayed a predominance of vestibular signs, including a head tilt (Table 1). The second cat, with a lesion in the territory of the proximal perforating artery at the level of the interthalamic adhesion, was obtunded. The cat circled ipsilateral to the lesion and had contralateral postural reaction deficits (Table 1). Given the low numbers of cats with presumptive thalamic CVAs it is not possible to determine if the recognised clinical syndromes noted in dogs can also be applied to cats.

In dogs, there was no observable statistical correlation between the site of CVA and a specific medical condition, although a single study of dogs with ischaemic CVAs revealed that 50% had concurrent systemic disease (6). The predominant medical conditions in these dogs were chronic kidney disease (CKD) and untreated hyperadrenocortism, affecting eight (44%) and six (33%) of the 18 dogs respectively. None of the cats in the present report were diagnosed with renal disease, which is particularly noteworthy given the high general prevalence of renal disease in cats, particularly in the ageing feline population.
It is difficult to draw reliable conclusions regarding disease prevalence between dogs and cats given the small referral cohort included. However, it is worthy to note that cats and dogs with presumed CVAs tend to present at an older age (the median age of cats included in this study was 12 years of age whilst the median age of dogs was 8 years (5)).

Pre-existing systemic disease was common in these cats presenting with CVAs. With 8/9 cats presenting with concurrent systemic disease, this appears more common than previously reported in dogs (50%). However, it is worthy to note that in two cats, HCM was mild with

no left atrial enlargement and is therefore unlikely to predispose to thromboembolic disease. Two cats with cerebellar CVAs have been previously described ante-mortem (9). Interestingly, both cats had concurrent medical conditions that may increase the risk of thromboembolic disease, specifically restrictive cardiomyopathy with valvular endocarditis and pulmonary neoplasia (9). In a separate study conducted post mortem, extra-cranial pathology was identified in five of seven cats with ischaemic CVAs, including hypertrophic cardiomyopathy and lymphoma (10). Other studies have shown that *Cuterebra* migration is associated with cerebrovascular disease in cats, although pathology was not restricted to ischaemic CVA alone (36, 37). Therefore, a cat presenting with signs consistent with a CVA warrants a full systemic investigation Most cats (8/9) survived to 48 hours following diagnosis and therefore had a good short-term outcome . As mentioned one cat was euthanized due to the severity of clinical signs, post-mortem examination in this case revealed an extensive CVA, in the territory of the rostral cerebellar artery in addition to moderate HCM, pulmonary adenocarcinoma and evidence of infarcts in the kidneys suggestive of a increased likelihood of thromboembolism. A further cat was euthanized (after an initial improvement in clinical signs) due to the severity of the concurrent medical condition (pulmonary mass with metastases) despite clinical improvement in neurological signs. These data suggest that short-term prognosis is good if the cat is neither severely affected nor impacted by critical concurrent disease. Previous reports in dogs have shown that prognosis is good if no underlying disease is found. In a single study with a total of 22 dogs with presumed CVAs, 23% died within 30 days, however, those that survived this period had a median survival time of 505 days (11). The low number of cases and the retrospective nature of the study make it impossible to determine long-term prognosis in cats with CVAs. In the cases observed in this study, follow-up information for long term

outcome was only available in two of the seven cats that survived to discharge; of those two, one had a good and the other an excellent outcome at a minimum of five weeks, although information beyond this time period is only sporadically available and conclusions cannot be drawn. This highlights a further limitation of the study due to its retrospective nature. It should be noted that all the cats included had neurological signs that improved prior to discharge. These inclusion criteria may inadvertently introduce bias in selecting for less severe cases, which may serve to exclude cats with extensive lesions or severe disease requiring more time before significant improvement is identified. Whilst it would be useful to document the long-term prognosis in these cases, the study period reviewed (11 years) and apparent low prevalence of ischaemic CVAs in cats means that further collection of long-term data would likely be problematic. Given the higher occurrence of concurrent disease identified in these cats, it is possible to argue that prognosis for CVAs is more guarded for the feline population in general, although this will be dependent on the severity and type of the relevant concurrent disease. A further study is necessary to determine the long-term outcome of these cases.

Conclusion

In conclusion, ischaemic CVAs appear to be an uncommon cause of neurological disease in cats. When they do occur, these data suggests they are most commonly located caudal to the tentorium, and in particular the cerebellum. All regions of the brain, however, may be affected and therefore vascular disease needs to be considered in any cat presenting with peracute onset non-progressive (over 24 hours) intracranial signs. Concurrent systemic disease in these cats was common and in particular HCM with or without concurrent

systemic hypertension. Despite this, the chance of recovery to discharge is favourable assuming neither the clinical presentation nor concurrent disease is severe.

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