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Thoracic vertebral canal stenosis in cats: clinical features, diagnostic imaging findings, treatment and outcome

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Abstract:	Objectives: Describe the clinical features, diagnostic imaging findings, treatment and outcome in cats with thoracic vertebral canal stenosis (TVCS). Methods: Medical records and imaging studies of cats with TVCS were retrospectively reviewed. Outcome was acquired from patient records and from owners or referring veterinary surgeons via a telephone questionnaire. For each case, breed, age and gender matched controls were identified with CT imaging of the thoracic vertebral column. For each cat, vertebral canal height was determined at three levels for each thoracic vertebra. Vertebral canal heights were compared between control cats of different breeds and between affected and control cats of the same breed. Results: Nine TVCS cases were included. British Shorthairs and male neutered cats were over-represented ($P<0.05$). Median age at presentation was 9 years. All cats presented for a chronic, progressive, painful, ambulatory, T3–L3 myelopathy. Five cats were treated conservatively, three surgically and one euthanized. Two cats treated surgically demonstrated improvement of clinical signs and one demonstrated initial improvement followed by deterioration. Of the conservatively treated cats, three deteriorated and two improved. Compared to controls, affected cats had a lower vertebral canal height at multiple thoracic vertebral levels, being most prominent for British Shorthairs and Domestic Shorthairs ($P<0.05$). Unaffected British Shorthairs had a lower thoracic vertebral canal height at multiple levels compared to control Domestic Shorthairs ($P<0.05$). Conclusions and relevance: TVCS should be considered a differential diagnosis in middle-aged to older cats presenting with a chronic, progressive, painful, T3-L3 myelopathy. The predisposition of British Shorthairs could be explained by a narrower vertebral canal in this breed.

Thoracic vertebral canal stenosis in cats: clinical features, diagnostic imaging findings, treatment and outcome

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ABSTRACT

Objectives: Describe the clinical features, diagnostic imaging findings, treatment and outcome in cats with thoracic vertebral canal stenosis (TVCS).

Methods: Medical records and imaging studies of cats with TVCS were retrospectively reviewed. Outcome was acquired from patient records and from owners or referring veterinary surgeons via a telephone questionnaire. For each case, breed, age and gender matched controls were identified with CT imaging of the thoracic vertebral column. For each cat, vertebral canal height was determined at three levels for each thoracic vertebra. Vertebral canal heights were compared between control cats of different breeds and between affected and control cats of the same breed.

Results: Nine TVCS cases were included. British Shorthairs and male neutered cats were over-represented ($P<0.05$). Median age at presentation was 9 years. All cats presented for a chronic, progressive, painful, ambulatory, T3–L3 myelopathy. Five cats were treated conservatively, three surgically and one euthanized. Two cats treated surgically demonstrated improvement of clinical signs and one demonstrated initial improvement followed by deterioration. Of the conservatively treated cats, three deteriorated and two improved. Compared to controls, affected cats had a lower vertebral canal height at multiple thoracic vertebral levels, being most prominent for British Shorthairs and Domestic Shorthairs ($P<0.05$). Unaffected British Shorthairs

had a lower thoracic vertebral canal height at multiple levels compared to control Domestic Shorthairs ($P<0.05$).

Conclusions and relevance: TVCS should be considered a differential diagnosis in middle-aged to older cats presenting with a chronic, progressive, painful, T3-L3 myelopathy. The predisposition of British Shorthairs could be explained by a narrower vertebral canal in this breed.

INTRODUCTION

Over recent years, our knowledge of feline spinal disease has increased with more patients undergoing advanced diagnostic investigations and imaging procedures, such as magnetic resonance imaging (MRI). Infectious and neoplastic disorders, such as feline infectious peritonitis (FIP) and lymphoma have previously been considered to represent the most common feline spinal disorders.[1,2] However, a more recent study indicated that non-lymphoid neoplasia and intervertebral disc disease were also common causes of spinal disease in cats.[3] Thoracic vertebral canal stenosis (TVCS) was recently documented to be amongst the ten most common causes of spinal disease in the cat[3] and a recent report described the successful surgical management of two cats with articular process hypertrophy causing TVCS.[4] There is however little known about the clinical presentation, imaging findings and outcome of cats with this disease.

Vertebral canal stenosis is an abnormal narrowing of the vertebral canal, resulting in compression of the spinal cord or nerve roots. It can occur focally, segmentally or generalised throughout the vertebral column and can be classified by aetiology into congenital, developmental and acquired causes.[5] Congenital stenosis results from malformations present at birth while developmental stenosis is caused by an active underlying process that remains present through the growth period, until the vertebrae reach maturity. Acquired stenosis may result from a variety of pathologies such as hypertrophied ligaments, intervertebral disc herniation, and degenerative articular

changes. Vertebral canal stenosis can further be classified into absolute and relative vertebral canal stenosis. A vertebral canal diameter that results in compression of neural elements directly is termed absolute stenosis, whereas a diameter that is less than normal but not causing compression of neural elements is termed relative stenosis.[6] The latter condition results in decreased available space for the spinal cord to compensate for extradural space occupying conditions. Relative vertebral canal stenosis therefore predisposes animals to develop clinical signs when relatively mild space-occupying pathologies, such as age-related intervertebral disc protrusion or ligamentous hypertrophy occur.[5,7]

Several studies have reported the clinical characteristics and treatment of TVCS in dogs.[8-11] However, there is a paucity of information about this disease in feline patients. The aims of this study were therefore to describe the clinical and diagnostic imaging characteristics, treatment and outcome in a group of cats with TVCS. We hypothesised that cats diagnosed with TVCS would demonstrate relative vertebral canal stenosis. Furthermore, it was hypothesised these cats would have a characteristic clinical presentation and would demonstrate a favourable response to surgical management.

MATERIALS AND METHODS

Ethics approval was granted by the Royal Veterinary College (RVC) Ethics and Welfare committee (reference number: SR2018-1654).

Criteria for inclusion

The digital medical records of the University of London, Royal Veterinary College (RVC), Small Animal Referral Hospital from January 1, 2010, through September 1, 2018, were reviewed to identify cats with a diagnosis of TVCS. For inclusion in the study, cats were required to have MRI and neurological examination findings consistent with a diagnosis of TVCS and available follow-up information. A diagnosis of TVCS was made based on the findings of reduced dimensions of the thoracic vertebral canal with associated compression of the spinal cord with or without adjacent spinal cord intraparenchymal signal intensity (ISI) changes. Cats were not included if a clear degenerative or anatomical change was present that would likely cause clinical signs in the absence of reduced vertebral canal size. All diagnostic imaging studies and medical records were reviewed by a board-certified veterinary neurologist (SDD) to evaluate study eligibility. Information recorded for each cat included signalment, duration and type of clinical signs, treatment prior to referral and response, general physical and neurological examination findings. Diagnostic tests performed and results, treatment received, duration of hospitalisation and presence of complications were also recorded.

Cats were excluded if the medical files or imaging records were not available or if other abnormalities were detected that could have caused or contributed to the cat's clinical signs.

For comparative reasons, a control group was established. For each cat identified with a diagnosis of TVCS it was aimed to include 10 breed and gender matched controls. For inclusion, control patients were to be skeletally mature and have undergone full computed tomography (CT) imaging of the thoracic vertebral column for reasons unrelated to gait abnormalities, spinal disease or trauma. The signalment of each cat and reason for CT imaging were recorded from the medical records.

Diagnostic imaging

For all included cats with a diagnosis of TVCS, MRI was performed using a 1.5T unit (Intera; Philips Medical Systems). MRI was performed under general anaesthesia and included a minimum of T2-weighted (T2W) and T1-weighted (T1W) sagittal and transverse images. T1W sagittal and transverse images were acquired after administration of gadolinium-based contrast medium (0.5ml/kg, IV). For each cat, the site(s) and suspected anatomical cause of TVCS were recorded as well as any associated ISI changes relative to normal spinal cord parenchyma. ISI changes were recorded if a T2-weighted hyperintensity was seen, with or without a corresponding hypointense ISI

change on T1-weighted images. For all included case-controls, CT imaging was performed with a 16-slice helical CT scanner (PQ 500, Universal Systems, Solon; GE Healthcare), under sedation or general anaesthesia. Sagittal reconstructions were made after the transverse images were acquired.

Measurements

All CT and MRI measurements were made by a veterinary neurology specialist-in-training (SG) under the supervision of a board-certified veterinary neurologist (SDD) on a commercially available DICOM viewing software (Horos, version 1.1.7, www.horosproject.org). For cats with a diagnosis of TVCS, measurements were made on mid-sagittal T1W MR images. For the control group, measurements were made on mid-sagittal CT images of the thoracic vertebral canal in the bone window (helical scan mode, slice thickness 2mm, interslice interval 1mm, collimation pitch 16 x 1.5mm, 120kVp, 100mA and a 512 x 512 matrix). The accuracy of the digital measurement tool was limited to 0.01mm. In each cat, for vertebrae T1 through T13, vertebral canal height was determined at the cranial (VCHcr), middle (VCHm) and caudal (VCHcd) aspects of the vertebral body as previously described.[12] To improve visualisation of the dorsal margin of the vertebral body, a reference line was drawn to connect the most craniodorsal and most caudodorsal points. Vertebral canal height measurements were made perpendicular to this reference line. The VCHcr was measured from the most craniodorsal point of the

vertebral body to the lamina. The VCHm was measured from the point that corresponded to half the length of the vertebral body to the lamina. The VCHcd was measured from the most caudodorsal point of the vertebral body to the lamina.

Outcome assessment

Outcome information for cats with a diagnosis of TVCS was acquired from a combination of: medical records of re-examination visits at the RVC Small Animal Referral Hospital, telephone interviews with referring veterinarians and owners. Referring veterinarians were first contacted and asked a series of questions regarding the patient's clinical status, current medications, neurologic deficits present, and progression after commencement of treatment. For cases that were deceased, the date, cause of death, and last documented neurologic status were recorded. Conforming to local ethics and welfare guidelines, the owners of cats known to have died were not contacted further. Owners of cats last known to be alive were mailed a letter that included the study details and a standardized questionnaire that had been reviewed and approved by a local ethics and welfare committee. Telephone interviews were conducted with one investigator (SG) based on the questionnaire which included information on activity levels, paresis, incontinence, pain levels, type of treatment received, response to treatment and quality of life (supplementary material). Treatment outcome was defined as improved, stabilized or deteriorated based on change in the

original neurological signs. Improvement was defined as an increase in pelvic-limb function score without occurrence of urinary or faecal incontinence or pain. Deterioration was defined as a decrease in pelvic-limb function score, occurrence of urinary or faecal incontinence or continued pain.

Statistical analysis

Data analysis was performed using a statistical software package (SPSS Statistics for OSx, Version 24.0, IBM Corp, Armonk, NY). Data were tested for normal distribution using Kolmogorov-Smirnov test and presented as mean +/- standard deviation (SD). Independent T-tests were used to compare the cranial, middle and caudal vertebral canal heights of each vertebra between unaffected cats of different breeds and between affected and control cats of the same breed. An X^2 test was used to compare the prevalence of sex and breeds that were included more than twice in the list of affected breeds (Domestic Shorthair, British Shorthair) to the general hospital population seen over the same period. For all comparisons, values of $P < 0.05$ were considered significant.

RESULTS

Nine cats with TVCS were included in the study and 81 control animals.

Of the 12 cats with TVCS identified from the database, three cats were excluded due to incomplete medical or imaging records. The nine cats with TVCS included in the study consisted of three British Shorthairs, three Domestic Shorthairs and one each of Bengal, Exotic Shorthair and Maine Coon. Eight of the cats were male neutered and one cat was female neutered. Median age was 9.0 years (range, 5.0 years – 14.0 years). Compared to the general hospital population, British Shorthairs (P=<0.0001) and male neutered cats (P=0.017) were significantly overrepresented.

Eighty-one control cats were included, comprising 30 British Shorthairs, 30 Domestic Shorthairs, 10 Bengals, five Exotic Shorthairs and six Maine Coons. For Exotic Shorthair and Maine Coon breeds, five and six control cases were included respectively, due to insufficient CT studies being available. Ten female neutered and 71 male neutered cats were included with a median age of 8 years (range, 1.2 years – 16.8 years). Control cats underwent CT for variety of reasons including respiratory disease (n=20), abdominal disease (n=14), neoplastic disease (n=40) and further investigation of immune mediated disease (n=7).

Clinical presentation and diagnostic findings in cats with TVCS

Duration of clinical signs prior to presentation ranged between 1 day and 9 months with eight cats demonstrating progressive clinical signs of at least 2 weeks duration. One cat demonstrated an acute onset of clinical signs following a minor trauma. All cats demonstrated ambulatory paraparesis and proprioceptive ataxia in the pelvic limbs, lateralising in four cases. All cats demonstrated hyperaesthesia on spinal palpation and none had a history of faecal or urinary incontinence. Neuroanatomical localisation was to the T3-L3 spinal cord segments in all cases.

Imaging

MRI studies demonstrated a single site of TVCS in eight cats and at three sites in one cat. In all cases, the thoracic vertebral canal was subjectively narrowed with secondary, mild changes to adjacent structures contributing to stenosis. In six cases (and at eight locations), vertebral canal stenosis was secondary to a combination of ventral spinal cord compression due to mild intervertebral disc protrusion and dorsal compression caused by the dorsal lamina and ligamentum flavum (n=4) or articular processes (n=2). The T3-T4 and T11-T12 intervertebral disc spaces were most often affected (n=3 sites of spinal cord compression for each), followed by T4-T5 (n=1) and T8-T9 (n=1). In two cases, stenosis was secondary to mild hypertrophy of the dorsal lamina and ligamentum flavum alone,

196 at T2 and T5. In one case, marked dorsoventral compression of the spinal cord was
197 present at T9 secondary to a subjectively narrowed vertebral canal, in the absence of an
198 appreciable anatomical abnormality. Intraparenchymal signal intensity changes at the site
199 of spinal cord compression were characterized by an ill-defined, T2W hyperintense and
200 T1W isointense lesion in eight cats and a focal T2W hyperintense and T1W hypointense
201 lesion in one cat. Abnormal contrast uptake was not noted in any cat. In all cases,
202 intervertebral disc protrusion was mild and anatomical changes to the vertebrae or
203 associated structures were difficult to appreciate despite obvious spinal cord compression
204 and adjacent intra-parenchymal signal intensity changes (Figure 1). Additional CT studies
205 were available for two cats and demonstrated no obvious anatomical abnormalities
206 leading to TVCS.

207 *Ancillary diagnostics*

208 Haematology and serum biochemistry results were available for all 9 cats and were
209 unremarkable in all cases. FIV and FeLV status were available for 4 cats, all of which
210 were negative. Toxoplasma serology was performed in 3 cases and was negative in all.
211 Four cats had cerebrospinal fluid (CSF) analysis performed from the lumbar cistern. Total
212 nucleated cell count (TNCC) and total protein (TP) concentrations were considered
213 normal in three cases (TNCC <5mm^3 and TP <0.45g/l). In one case, there was evidence
214 of mild albuminocytologic dissociation with a total protein concentration of 0.75g/l

(reference, <0.45g/l). In one cat, toxoplasma and feline coronavirus PCRs performed on CSF were negative.

Treatment

Owners were informed of treatment options by a neurology specialist or specialist in training and treatment undertaken was based on owner preference. In one cat presenting acutely, medical management was preferentially recommended. Eight cats underwent medical (n=5) or surgical (n=3) treatment of the condition. One cat was euthanized shortly following diagnosis due to deteriorating neurological signs after which the owners declined further treatment.

Surgical treatment

Two cats underwent a left sided hemilaminectomy for removal of an articular facet joint perceived to be contributing to vertebral canal stenosis. The procedure was performed at the T11-T12 and T3-T4 intervertebral disc spaces respectively. These two cats demonstrated no post-operative deterioration and were ambulatory at the time of discharge. One cat with stenosis at T4-T5 underwent initial medical management with meloxicam, gabapentin and restricted exercise. Due to continued progression of paraparesis and proprioceptive ataxia over 4 weeks, a T3 to T5 dorsal laminectomy was performed. Surgery revealed a subjectively narrow vertebral canal without obvious

anatomical abnormalities. This cat demonstrated deterioration post-operatively and was non-ambulatory at the time of discharge with good pelvic limb movement. All cats were hospitalised for 6 days post-surgery and were discharged with instructions for 4-weeks restricted exercise and meloxicam (0.1mg/kg, PO, q24h) for 7-14 days. At re-check examinations between 1 and 2 months following surgery, all cats that underwent surgical treatment were ambulatory and comfortable with a mild improvement in neurological function compared to original presentation. None of the cats were considered neurologically normal.

Follow-up information was available for all three cats treated surgically. Two cats were alive at the time of follow up (11.5 months and 74.3 months). One cat underwent a hemilaminectomy and was considered neurologically normal 74.3 months after surgery. The other cat underwent a dorsal laminectomy and had serial neurological examinations every 2 to 3 months at the study institution. It demonstrated signs of slow deterioration (increased paraparesis and faecal incontinence) approximately 8 months following surgery, after an initial period of improvement. The MRI study at the time of diagnosis demonstrated a combination of intraspinal T2W hyperintensity and T1W hypointensity at the site of spinal cord compression (Figure 2). The third cat had been euthanized 51 months after hemilaminectomy surgery (at 14 years of age) due to the development of non-ambulatory paraparesis. This cat demonstrated an initial, sustained improvement in

neurological signs and lived with a mild paraparesis and proprioceptive ataxia for approximately 4 years, prior to a chronic deterioration. Unfortunately, neither cat demonstrating a period of stabilisation and subsequent deterioration underwent further investigations and the exact cause of neurologic deterioration remains unknown.

Medical Treatment

Three cats received meloxicam (0.05mg/kg, PO, q24h) alone and one cat received a combination of meloxicam and gabapentin (10mg/kg, PO, q8h). One cat was started on oral prednisolone at an initial dose of 0.6mg/kg, PO, q24h, tapering down over a period of 3 months.

Follow-up information was available for all five cats treated medically. One cat was alive at the time of follow up (32 months) and had been tapered off oral medication (prednisolone). This cat was reported to be neurologically improved but remained with a mild ambulatory paraparesis and proprioceptive ataxia. One cat demonstrated improvement and was reported to be neurologically normal before death in a road traffic accident 15 months after treatment. This cat had demonstrated an acute onset of clinical signs following a minor trauma. Three cats had been euthanased at 1, 35 and 48.5 months after starting treatment due to deteriorating paraparesis, with one cat becoming non-ambulatory. One of these three cats also developed urinary and faecal incontinence.

Vertebral Canal Measurements

One MRI study was of insufficient quality for accurate measurements to be performed. Therefore, imaging studies from eight cats with TVCS were further evaluated.

Unaffected British Shorthairs were found to have a significantly smaller cranial (VCHcr), middle (VCHm) and/or caudal (VCHcd) thoracic vertebral canal heights at all levels when compared to Domestic Shorthairs ($P<0.05$). More specifically, at T4, T5, T9 and T11 VCHcr, VCHm and VCHcd were all significantly smaller in British Shorthairs compared to Domestic Shorthairs (Table 1). Unaffected Bengals demonstrated a significantly smaller cranial and middle sagittal vertebral canal height at T5 and unaffected Maine Coons did not demonstrate a significantly different vertebral canal height at any level when compared to Domestic Shorthairs.

When compared to controls, cats with TVCS had a significantly smaller thoracic vertebral canal heights at multiple levels. This was most notable in the Domestic Shorthair and British Shorthair breeds ($P<0.05$, Table 2). More specifically, at T2 to T7, T11 and T12, VCHcr, VCHm and/or VCHcd were significantly smaller in affected British Shorthairs compared to control British Shorthairs. At T1, T3, T5 and T8 to T13, VCHcr, VCHm and/or VCHcd were significantly smaller in affected Domestic

Shorthairs compared to control Domestic Shorthairs. VCHcr was significantly smaller at T9 in affected Bengals compared to control Bengals and VCHca was significantly smaller at T3 in affected Maine Coons compared to control Maine Coons.

Table 1. Mean values of cranial (VCHcr), middle (VCHm) and caudal (VCHcd) vertebral canal heights (mm) in control British Shorthair (BSH) and control Domestic Shorthair (DSH) cats.

Vertebra	Mean VCHcr		Mean VCHm		Mean VCHcd	
	BSH	DSH	BSH	DSH	BSH	DSH
1	6.51	6.80	5.68	5.92	6.13*	6.56*
2	5.72	6.23	4.96*	5.20*	5.36	5.52
3	5.14	5.18	4.58*	4.78*	5.25*	5.58*
4	5.13*	5.31*	4.54*	4.78*	5.22*	5.61*
5	5.17*	5.54*	4.60*	4.82*	5.29*	5.55*
6	5.24*	5.49*	4.54	4.72	5.32	5.49
7	5.37	5.52	4.54*	4.80*	5.31	5.53
8	5.28*	5.52*	4.52	4.72	5.10*	5.41*
9	5.16*	5.43*	4.43*	4.68*	4.90*	5.16*
10	5.00	5.26	4.32	4.33	5.19*	5.62*
11	5.04*	5.59*	4.22*	4.50*	4.94*	5.57*
12	4.46*	5.21*	4.40	4.56	5.13*	5.59*
13	4.44*	4.92*	4.60	4.79	5.19*	5.64*

*Signifies a significant difference between values in British Shorthair and Domestic Shorthair cats ($P<0.05$).

Table 2. Mean values of cranial (VCHcr), middle (VCHm) and caudal (VCHcd) vertebral canal heights (mm) in affected British Shorthair (BSHa) and control British

Shorthair (BSHc) cats. BSHa measurements acquired from MRI images and BSHc measurements from CT images.

Vertebra	Mean VCHcr		Mean VCHm		Mean VCHcd	
	BSHc	BSHa	BSHc	BSHa	BSHc	BSHa
1	6.51	5.67	5.68	5.38	6.13	5.37
2	5.72	5.04	4.96	4.59	5.36*	4.39*
3	5.14	4.53	4.58*	3.94*	5.25*	4.00*
4	5.13*	3.78*	4.54*	3.73*	5.22*	4.11*
5	5.17*	3.92*	4.60*	3.78*	5.29*	4.10*
6	5.24*	4.05*	4.54*	3.64*	5.32*	3.75*
7	5.37*	4.41*	4.54	4.41	5.31*	4.22*
8	5.28	4.61	4.52	4.50	5.10	4.50
9	5.16	4.58	4.43	4.55	4.90	4.43
10	5.00	4.42	4.32	4.46	5.19	4.43
11	5.04	4.44	4.22	4.32	4.94	4.77
12	4.46	4.67	4.40	4.39	5.13	4.54
13	4.44	4.49	4.60	4.65	5.19	4.30

*Signifies a significant difference between values in affected and control cats ($P<0.05$).

DISCUSSION

Thoracic vertebral canal stenosis is poorly characterised despite being a common cause of spinal disease in the cat.[3] This study demonstrated TVCS to be most frequent in middle aged to older male neutered cats presenting with a chronic, progressive, painful, T3-L3 myelopathy. Imaging studies typically demonstrated dorsoventral spinal cord compression and ISI changes in the absence of a marked anatomical abnormality. Surgical and medical treatment of this disease appears to carry a variable prognosis.

In dogs, TVCS typically occurs in the cranial thoracic segments of young, large breed dogs with a conformation characteristic of Molosser breeds. Lateral and dorsolateral spinal cord compression results from enlargement and malformation of the articular facet joints, with a developmental aetiology considered likely.[8-11] In contrast, TVCS in cats represents a different disease process. In this study, all cats were middle aged to older at presentation and typically presented with chronic, progressive clinical signs. Imaging studies demonstrated no evidence of marked anatomical abnormality to the vertebrae or associated structures despite obvious spinal cord compression and adjacent ISI changes. Any changes seen at the site of compression were considered mild, expected to be age related and would not be anticipated to cause clinical signs in the presence of normal vertebral canal dimensions. Given the clinical presentation and imaging findings, we hypothesised that these cats may have a preexisting relative

vertebral canal stenosis. Reduced 'free space' for the spinal cord may then lead to the development of an absolute stenosis and clinical signs secondary to mild age-related degenerative changes, such as intervertebral disc protrusion, ligamentous hypertrophy or articular process hypertrophy.

A recent case report described thoracic vertebral canal stenosis in two cats, secondary to bilateral articular process hypertrophy.[4] It is unclear if this report describes a different disease process to that seen in the current study, given the appreciable articular process hypertrophy on imaging. However, in agreement with the current study, both cats were middle aged or older at presentation and demonstrated a chronic, progressive T3-L3 myelopathy. In contrast to the present study, both cats had evidence of ventral spondylosis deformans at the site of vertebral stenosis, suggesting chronic vertebral instability and secondary articular process hypertrophy as the pathophysiology.[4]

Overall, six of the nine cats affected by TVCS in the present study were of purebred descent and results suggested a breed related predisposition in British Shorthairs. Interestingly, this breed was previously reported to be more commonly diagnosed with thoracolumbar intervertebral disc disease.[13] Although it is unclear why British Shorthairs may be predisposed to both TVCS and thoracolumbar intervertebral disc

disease, it is possible that pre-existing relative vertebral canal stenosis contributes to the development of clinical signs with age-related degenerative changes.

The most frequent sites of stenosis were the T3-T4 and T11-T12 intervertebral disc spaces, consistent with the sites affected in the aforementioned case report.[4] It is unclear why the sites of stenosis varied and it may be expected for degenerative changes to primarily occur in the caudal thoracic vertebral column, which shows a higher degree of flexibility and is subject to increased biomechanical forces compared with the cranial thoracic compartment.[14,15] Equally, it is possible relative stenosis is limited to selected regions of the vertebral canal.

Susceptibility of the spinal cord to compression depends on vertebral canal dimensions in both the transverse and sagittal planes.[16] This data should thus be interpreted with caution given measurements were only acquired in the sagittal plane. However, our results suggest that British Shorthair cats without TVCS have a relatively smaller thoracic vertebral canal height at multiple levels compared to Domestic Shorthair cats without TVCS. This might provide an explanation for a predisposition to development of TVCS in this breed. Correspondingly, TVCS affected cats demonstrated a significantly lower vertebral canal height at multiple thoracic levels, reflecting the sites of stenosis on MRI studies.

Although well established in dogs,[7,17-19] there is little known about normal vertebral canal dimensions in cats. Breed and body size are known to influence morphometric dimensions of the vertebral column in dogs. Large breed dogs and Dachshunds have lower vertebral canal dimensions than small breeds at multiple thoracic and lumbar vertebral levels. Sites of reduced vertebral canal dimensions appear consistent with those sites most commonly clinically affected by spinal cord compression.[17] Similarly, it may be speculated that cats affected by TVCS have lower thoracic vertebral canal dimensions than normal. However, further morphometric studies are required in cats to determine normal vertebral canal dimensions and possible breed or size-related variations, as seen in dogs.

There is little information available detailing treatment and prognosis for TVCS in cats. A good outcome 6-months after surgery has been reported for two cats with TVCS secondary to articular process hypertrophy.[4] Similarly, of the three cats that underwent surgery in the present study, all demonstrated a good outcome 1-2 months post-surgery and two demonstrated a good long-term outcome.

The cat that underwent a T3-T5 dorsal laminectomy demonstrated a progressive deterioration 8 months following surgery. This cat was initially treated medically

without success and demonstrated a focal T2W hyperintense and T1W hypointense intraparenchymal lesion at the site of spinal cord compression. These imaging changes may be considered consistent with a cystic or cavitary type lesion.[20] These parenchymal signal intensity changes have previously been associated with neurological deterioration and histopathological evidence of segmental chronic myelomalacia and gliosis in two dogs with disc associated cervical spondylomyelopathy.[21] Equally, there is growing evidence in people that these signal intensity changes represent pathologically irreversible spinal cord damage and have been associated with a poor prognosis in cases of cervical spondylotic myelopathy.[22-24] It is unknown why the cat in this report demonstrated progressive deterioration after initial clinical improvement was observed. Although it is possible that the specific intraparenchymal intensity changes seen at the time of diagnosis reflected irreversible spinal cord damage, it is also possible that the delay in surgical treatment associated with initial medical management affected prognosis. This cat did not undergo repeat imaging at the time of neurological deterioration. Therefore, it cannot be excluded that an additional site of spinal cord compression or another spinal problem occurred.

In three of the five cats treated with medical management alone, the condition continued to slowly progress to the point of euthanasia. This likely reflects the chronic, progressive nature of degenerative changes in the vertebral canal. Notably, the one cat

that presented acutely made a full and rapid recovery with medical management alone. It may be hypothesised that the already compromised spinal cord suffered an acute contusive injury in the region of stenosis.

The present study was limited by the small number of cats with TVCS and by its retrospective nature, which necessitated reliance on medical records accuracy and did not allow for standardized follow-up assessment. Not all control cats underwent neurological examination and thus mild spinal disease could not be completely excluded. Due to an inadequate number of CT studies in TVCS affected cats, MRI measurements were compared with CT measurements in control cats. Although this can be considered less accurate, a previous study demonstrated good agreement for vertebral canal height measurements between low field MRI and CT.[25] Equally, imaging measurements represented true measurements found on cadaveric specimens. In the present study, only sagittal vertebral canal measurements could be acquired due to lack of transverse studies of the entire thoracic vertebral column in TVCS affected cats. Linear ratios have previously been suggested unreliable in predicting relative vertebral canal stenosis in dogs[12,26] and people[27,28] and therefore absolute measurements were determined and compared. Relative vertebral canal stenosis depends on the transverse and sagittal vertebral canal dimensions, as well as the dimensions of the spinal cord.[12] Cross-sectional area measurements on transverse images may be

considered a more reliable determinant of relative vertebral canal stenosis in future studies.

CONCLUSIONS

TVCS should be considered a differential diagnosis in middle-aged to older cats presenting with a chronic, progressive, painful, T3-L3 myelopathy. Its prevalence appears to be higher in British Shorthairs and male neutered cats. Further studies are required to determine optimal treatment in these cats and it is possible, outcome may vary according to the anatomical structures contributing to stenosis. Medical management typically resulted in a slow progression of clinical signs. Surgical management resulted in a good short-term outcome and variable long-term outcome. It is suspected these cats may have a pre-existing relative stenosis of the thoracic vertebral canal and develop absolute stenosis secondary to age-related degenerative changes of the vertebral column and associated structures. It remains unknown if vertebral stenosis is generalised throughout the vertebral canal or limited to the thoracic region and equally, if there is variation between different breeds and size of cat. Although further studies are necessary, the predisposition of British Shorthairs could potentially be explained by a narrower vertebral canal in this breed compared to other breeds.

Supplementary material

The questionnaire used for conducting telephone interviews.

Conflict of interest

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Ethical approval

This work involved the use of non-experimental animal(s) only (owned or unowned), and followed established internationally recognised high standards ('best practice') of individual veterinary clinical patient care. Ethical approval from a committee was not necessarily required.

Informed Consent

Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work for the procedure(s) undertaken. No animals or humans are identifiable within this publication, and therefore additional Informed Consent for publication was not required.

REFERENCES

1 Gonçalves R, Platt SR, Llabrés-Díaz FJ, et al. **Clinical and magnetic resonance imaging findings in 92 cats with clinical signs of spinal cord disease**. *J Feline Med Surg* 2009;1:53–59.

2 Marioni-Henry K, Vite CH, Newton AL, et al. **Prevalence of disease of the spinal cord of cats**. *J Vet Intern Med* 2004;18:851–858.

3 Mella SL, Cardy TJA, Volk HA, et al**. Clinical reasoning in feline spinal disease: which combination of clinical information is useful?** *J Feline Med Surg* 2019; Epub ahead of print 28 June 2019. DOI:10.1177/1098612X19858447.

4 Carletti BE, Espadas I and Sanchez-Masin D. **Thoracic vertebral canal stenosis due to articular process hypertrophy in two cats treated by hemilaminectomy with partial osteotomy of the spinous process.** *J Feline Med Surg Open Reports* 2019; Epub ahead of print 19 June 2019. DOI:10.1177/2055116919863176.

5 Sturges BK and Westworth DR. **Congenital spinal malformations in small animals.** *Vet Clin North Am Small Anim Pract* 2010;40:951-981.

6 Bailey CS and Morgan JP. **Congenital spinal malformations.** *Vet Clin North Am Small Anim Pract* 1992;22:985-1015.

7 De Decker, Da Costa RC, Volk HA, et al. **Current insights and controversies in the pathogenesis and diagnosis of disc-associated cervical spondylomyelopathy**

in dogs. *Vet Rec* 2012;171:531-537.

8 Stalin CE, Pratt JN, Smith PM, et al. **Thoracic stenosis causing lateral compression of the spinal cord in two immature Dogues de Bordeaux.** *Vet Comp Orthop Traumat* 2009;22:59-62.

9 Johnson P, De Risio L, Sparkes A, et al. **Clinical, morphologic, and morphometric features of cranial thoracic spinal stenosis in large and giant breed dogs.** *Vet Radiol Ultrasound* 2012;53:524-34.

10 Miller A and Marchevsky A. **Cranial thoracic vertebral canal stenosis in three juvenile large-breed brachycephalic dogs treated by unilateral hemilaminectomy**. *Vet Comp Orthop Traumat* 2017;30:223-229.

11 Hecht S, Michaels J, Childers S, et al. **Cranial thoracic stenotic spondylomyelopathy in a young Rottweiler.** *Vet Rec Case Rep* 2017;5.

12 De Decker S, Gielen IMVL, Duchateau L, et al. **Magnetic resonance imaging vertebral canal and body ratios in Doberman Pinschers with and without disk-associated cervical spondylomyelopathy and clinically normal English Foxhounds.** *Am J Vet Res* 2011;72:1496-1504.

13 De Decker S, Warner AS and Volk HA. **Prevalence and breed predisposition for thoracolumbar intervertebral disc disease in cats**. *J Feline Med Surg* 2017;19: 419-423.

14 Bouma JL. **Congenital Malformations of Vertebral Articular Processes in Dogs.**

Vet Clin North Am Small Anim Pract 2016;46:307-326.

15 Evans HE. **The skeleton: The vertebral column.** In: Evans HE (ed). *Miller's Anatomy of the Dog*. 3rd ed. Philadelphia:WB Saunders, 1993, pp.166-181.

16 Prasad SS, O'Malley M, Caplan M, et al. **MRI measurements of the cervical spine and their correlation to Pavlov's ratio.** *Spine* 2003;28:1263-1268.

17 Breit S. **Osteological features in pure-bred dogs predisposing to thoracic or lumbar spinal cord stenosis.** *Res Vet Sci* 2002; 73:87-92.

18 Da Costa RC, Parent JM, Partlow G, et al. **Morphologic and morphometric magnetic resonance imaging features of Doberman Pinschers with and without clinical signs of cervical spondylomyelopathy**. Am J Vet Res 2006;67:1601-12.

19 Martin-Vaquero P, da Costa RC and Lima CGD. Cervical spondylomyelopathy in Great Danes: A magnetic resonance imaging morphometric study. Vet J 2014; 201:64-71.

20 Alisauskaite N, Spitzbarth I, Baumgartner W, et al. **Chronic post-traumatic intramedullary lesions in dogs, a translational model.** *PLoS ONE* 2017;12: e0187746.

21 De Decker S, Gielen IMVL, Duchateau L, et al. **Evolution of clinical signs and predictors of outcome after conservative medical treatment for disk-associated cervical spondylomyelopathy in dogs.** *J Am Vet Med Assoc 2012*;240:848-857.

22 Ohshio I, Hatayama A, Kaneda K, et al. **Correlation between histopathologic**

features and magnetic resonance images of spinal cord lesions. *Spine* 1993;18: 1140-1149.

23 Suri A, Chabbra RP, Mehta VS, et al. **Effect of intramedullary signal changes on the surgical outcome of patients with cervical spondylotic myelopathy.** *Spine J* 2003;3:33-45.

24 Morio Y, Teshima R, Nagashima H, et al. **Correlation between operative outcomes of cervical compression myelopathy and MRI of the spinal cord.** *Spine* 2001;26:1238-1245.

25 De Decker S, Gielen I, Duchateau L, et al. **Agreement and repeatability of linear vertebral body and canal measurements using computed tomography (CT) and low field magnetic resonance imaging (MRI).** *Vet Surg* 2010;39:28-34.

26 De Decker S, Saunders JH, Duchateau L, et al. **Radiographic vertebral canal and vertebral body ratios in Doberman Pinschers with and without clinical signs of caudal cervical spondylomyelopathy.** *Am J Vet Res* 2011; 72:958-966.

27 Blackley HR, Plank LD and Robertson PA. **Determining the sagittal dimensions of the canal of the cervical spine: The reliability of ratios of anatomical measurements.** *J Bone Joint Surg* 1999;81:110-112.

28 Lim JK and Wong HK. **Variation of the cervical spinal Torg ratio with gender and ethnicity.** *Spine J* 2004;4:396-401.

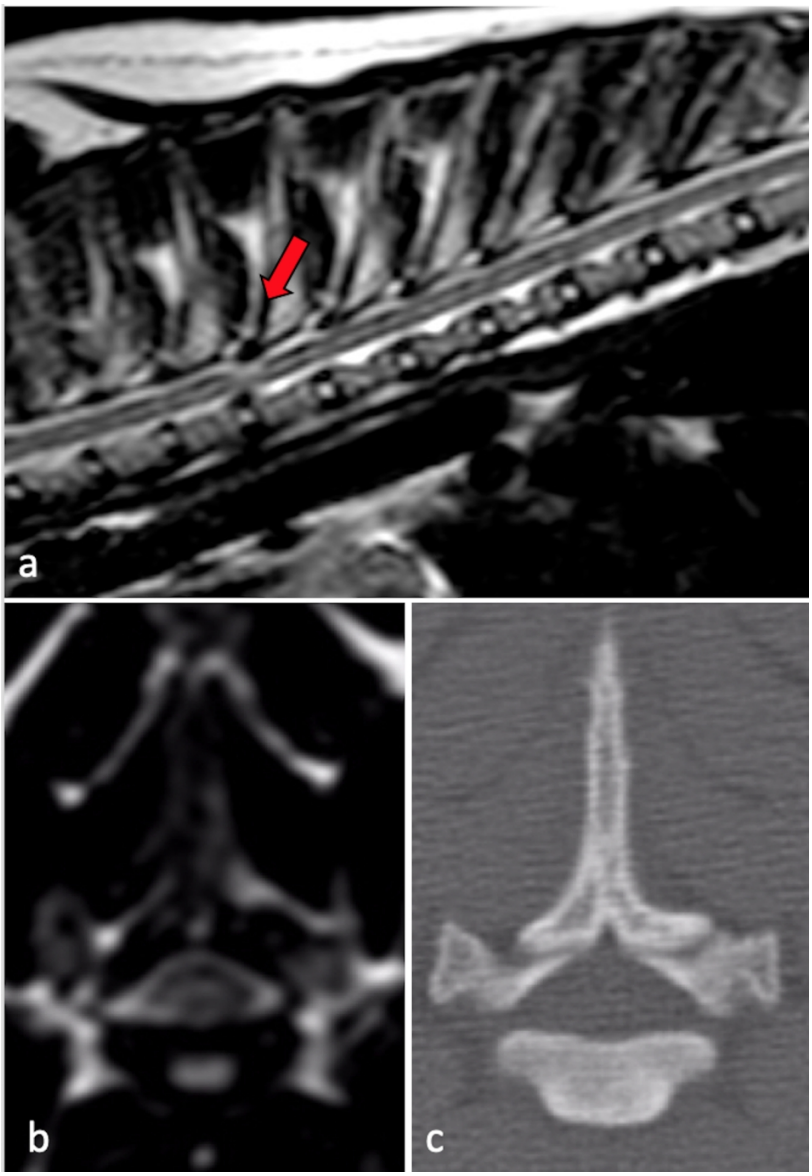

Figure 1 (a) T2W sagittal and (b) transverse MRI images of the thoracic spine at the level of T3-T4 (red arrow) demonstrating subjective thoracic vertebral canal narrowing, an intramedullary hyperintensity and (c) corresponding CT image at the same level demonstrating lack of obvious anatomical abnormality.

127x183mm (300 x 300 DPI)

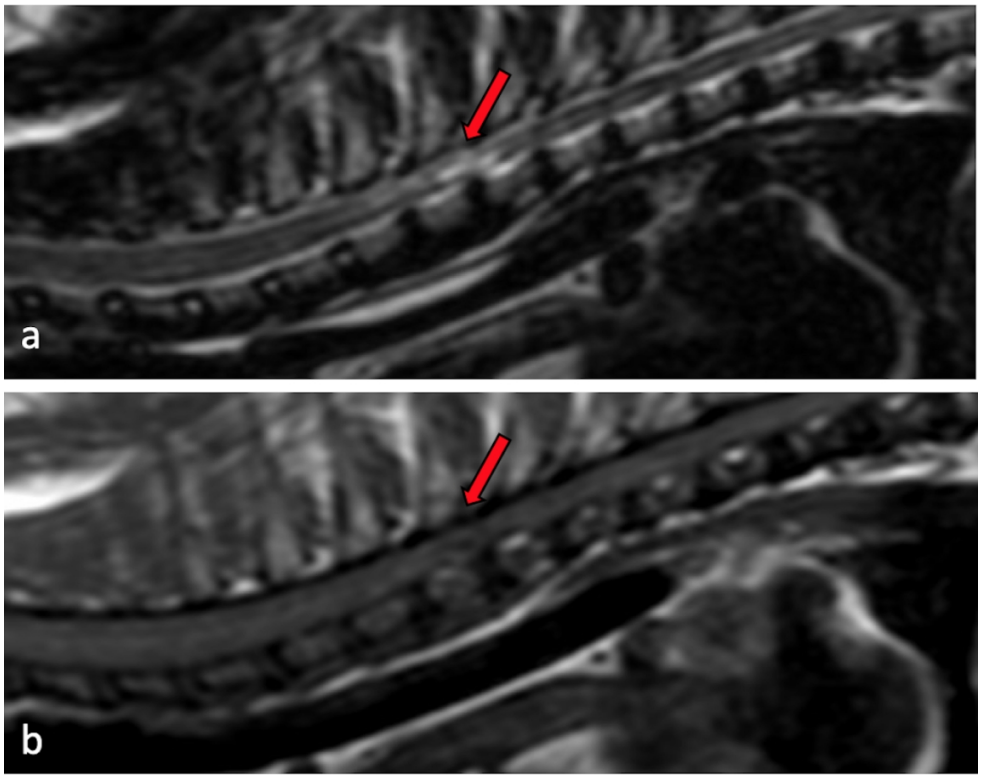

Figure 2 (a) T2W and (b) T1W sagittal images at the level of T4-T5 (red arrow) demonstrating a focal T2W hyperintense and T1W hypointense intraparenchymal lesion at the site of spinal cord compression. These imaging changes may be considered consistent with a cystic or cavitary type lesion.

106x84mm (300 x 300 DPI)

Questionnaire for owners of cats diagnosed with thoracic vertebral canal stenosis

1. How would you have rated your cats level of activity before the onset of clinical signs:

   1 2 3 4 5 6 7 8 9 10

   (1: could not have been less active; 10; could not have been more active)

2. How would you have rated your cats level of activity at the time of diagnosis:

   1 2 3 4 5 6 7 8 9 10

   (1: could not have been less active; 10; could not have been more active)

3. How would you rate your cats level of activity now:

   1 2 3 4 5 6 7 8 9 10

   (1: could not have been less active; 10; could not have been more active)

4. How would you have rated your cats function in their legs at the time of diagnosis:

   1 2 3 4 5 6 7 8 9 10

   (1: not able to walk without assistance; 10: completely normal)

5. How would you rate your cats function in their legs now:

   1 2 3 4 5 6 7 8 9 10

   (1: not able to walk without assistance; 10: completely normal)

6. Did your cat go outside before the onset of neurological signs:

   ☐ YES

   ☐ NO

7. Does your cat go outside now:

   ☐ YES

   ☐ NO

   If different from before the onset of neurological signs, why?

8. Is your cat able to the jump on furniture as they had prior to the onset of neurological signs?

   ☐ YES

   ☐ NO

9. How well do you perceive your cats control is over their urination?
   - ☐ A: completely normal – no leaking or accidents
   - ☐ B: moderate control - sometimes leaks or has accidents, but usually not
   - ☐ C: incontinent - most of the time my cat has no voluntary control over urination

10. How well do you perceive your cats control is over their defecation?
   - ☐ A: completely normal – no accidents
   - ☐ B: moderate control - sometimes there are accidents, but usually not
   - ☐ C: incontinent - most of the time my cat has no voluntary control over defecation

11. Did you perceive your cat to be painful following the onset of neurological signs?
   - ☐ YES
   - ☐ NO

12. Do you perceive your cat to be painful now?
   - ☐ YES
   - ☐ NO

   If YES, what signs of pain do you see?

13. Is your cat currently on medication? YES/NO
   - ☐ YES
   - ☐ NO

   If YES, what medication is he/she currently receiving? (please try and provide as much information as possible including name, how many times a day you give it)

14. Overall, do you feel your cat's clinical signs have improved, remained static or worsened since the start of treatment (supportive care, medical or surgical)?
   - ☐ IMPROVED
   - ☐ STATIC
   - ☐ WORSENED

15. If your cat is now normal how long did it take for them to return to normal?

16. If your cat improved but has not returned to normal then after how long did they stop showing signs of improvement?

17. What was your cats quality of life before the onset of the neurological signs:

1 2 3 4 5 6 7 8 9 10

(1: could not be worse; 10; could not be better)

18. How is your cats quality of life now:

1 2 3 4 5 6 7 8 9 10

(1: could not be worse; 10; could not be better)

Any other comments: