

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

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Abstract:	<p>Objectives: Describe the clinical features, diagnostic imaging findings, treatment and outcome in cats with thoracic vertebral canal stenosis (TVCS).</p> <p>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.</p> <p>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 ($P < 0.05$).</p> <p>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.</p>

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

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10 **ABSTRACT**

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

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

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

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

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

35 INTRODUCTION

36 Over recent years, our knowledge of feline spinal disease has increased with more
37 patients undergoing advanced diagnostic investigations and imaging procedures, such as
38 magnetic resonance imaging (MRI). Infectious and neoplastic disorders, such as feline
39 infectious peritonitis (FIP) and lymphoma have previously been considered to represent
40 the most common feline spinal disorders.^{1,2} However, a more recent study indicated that
41 non-lymphoid neoplasia and intervertebral disc disease were also common causes of
42 spinal disease in cats.³ Thoracic vertebral canal stenosis (TVCS) was recently
43 documented to be amongst the ten most common causes of spinal disease in the cat³ and
44 a recent report described the successful surgical management of two cats with articular
45 process hypertrophy causing TVCS.⁴ There is however little known about the clinical
46 presentation, imaging findings and outcome of cats with this disease.

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

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

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

71 **MATERIALS AND METHODS**

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

74 *Criteria for inclusion*

75 The digital medical records of the University of London, Royal Veterinary College
76 (RVC), Small Animal Referral Hospital from January 1, 2010, through September 1,
77 2018, were reviewed to identify cats with a diagnosis of TVCS. For inclusion in the
78 study, cats were required to have MRI and neurological examination findings consistent
79 with a diagnosis of TVCS and available follow-up information. A diagnosis of TVCS
80 was made based on the findings of reduced dimensions of the thoracic vertebral canal
81 with associated compression of the spinal cord with or without adjacent spinal cord
82 intraparenchymal signal intensity (ISI) changes. Cats were not included if a clear
83 degenerative or anatomical change was present that would likely cause clinical signs in
84 the absence of reduced vertebral canal size. All diagnostic imaging studies and medical
85 records were reviewed by a board-certified veterinary neurologist (SDD) to evaluate
86 study eligibility. Information recorded for each cat included signalment, duration and
87 type of clinical signs, treatment prior to referral and response, general physical and
88 neurological examination findings. Diagnostic tests performed and results, treatment
89 received, duration of hospitalisation and presence of complications were also recorded.

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

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

99 *Diagnostic imaging*

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

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

112 *Measurements*

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

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

130 *Outcome assessment*

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

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

150 *Statistical analysis*

151 Data analysis was performed using a statistical software package (SPSS Statistics for
152 OSx, Version 24.0, IBM Corp, Armonk, NY). Data were tested for normal distribution
153 using Kolmogorov-Smirnov test and presented as mean +/- standard deviation (SD).
154 Independent T-tests were used to compare the cranial, middle and caudal vertebral canal
155 heights of each vertebra between unaffected cats of different breeds and between
156 affected and control cats of the same breed. An X² test was used to compare the
157 prevalence of sex and breeds that were included more than twice in the list of affected
158 breeds (Domestic Shorthair, British Shorthair) to the general hospital population seen
159 over the same period. For all comparisons, values of $P < 0.05$ were considered
160 significant.

161 **RESULTS**

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

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

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

178 **Clinical presentation and diagnostic findings in cats with TVCS**

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

186 *Imaging*

187 MRI studies demonstrated a single site of TVCS in eight cats and at three sites in one cat.
188 In all cases, the thoracic vertebral canal was subjectively narrowed with secondary, mild
189 changes to adjacent structures contributing to stenosis. In six cases (and at eight
190 locations), vertebral canal stenosis was secondary to a combination of ventral spinal cord
191 compression due to mild intervertebral disc protrusion and dorsal compression caused by
192 the dorsal lamina and ligamentum flavum (n=4) or articular processes (n=2). The T3-T4
193 and T11-T12 intervertebral disc spaces were most often affected (n=3 sites of spinal cord
194 compression for each), followed by T4-T5 (n=1) and T8-T9 (n=1). In two cases, stenosis
195 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 <5mm³ 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

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

217 **Treatment**

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

224 *Surgical treatment*

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

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

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

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

256 *Medical Treatment*

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

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

270 **Vertebral Canal Measurements**

271 One MRI study was of insufficient quality for accurate measurements to be performed.

272 Therefore, imaging studies from eight cats with TVCS were further evaluated.

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

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

287 Shorthairs compared to control Domestic Shorthairs. VCHcr was significantly smaller
 288 at T9 in affected Bengals compared to control Bengals and VCHca was significantly
 289 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 ($P < 0.05$).

290 **DISCUSSION**

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

401 **CONCLUSIONS**

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

416 **Supplementary material**

417 The questionnaire used for conducting telephone interviews.

418 **Conflict of interest**

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425 **Ethical approval**

426 This work involved the use of non-experimental animal(s) only (owned or unowned),
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428 individual veterinary clinical patient care. Ethical approval from a committee was not
429 necessarily required.

430 **Informed Consent**

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

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For Peer Review

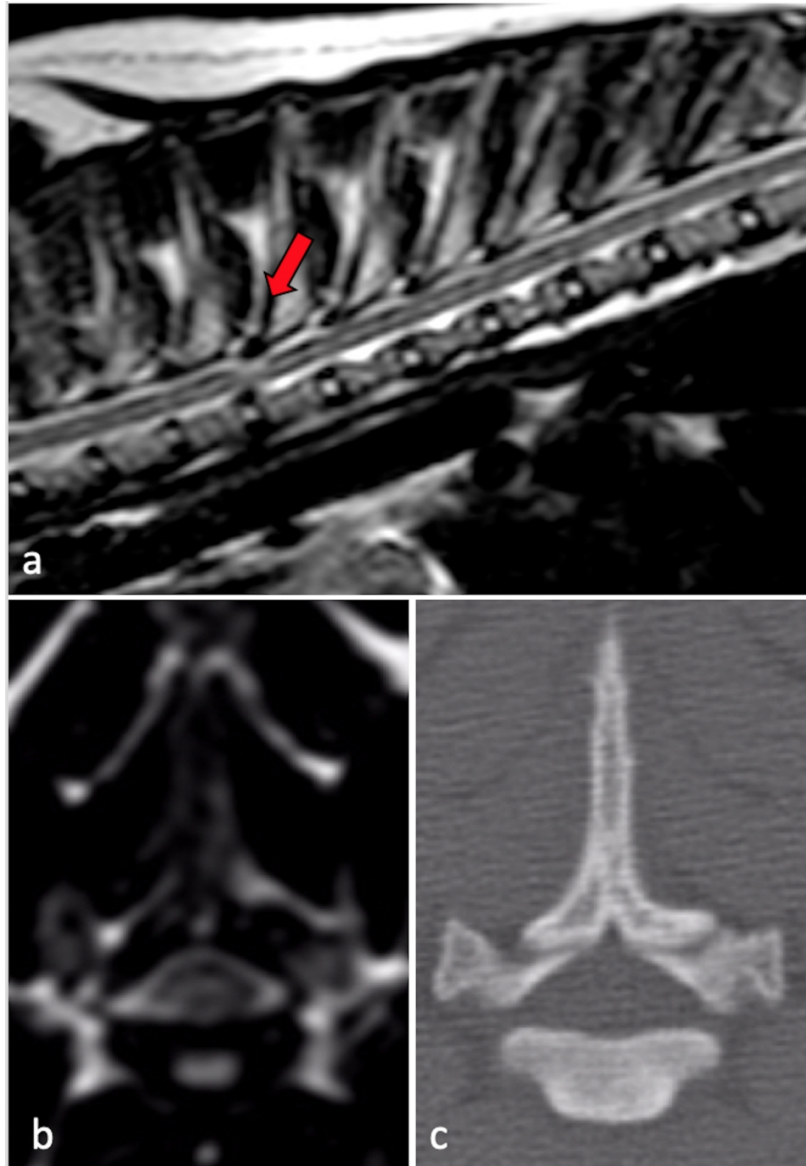


Figure 1 (a) T2W sagittal and (b) transverse MRI images of the thoracic spine at the level of T3-T4 (red arrow) demonstrating subjective thoracic vertebral canal narrowing, an intramedullary hyperintensity and (c) corresponding CT image at the same level demonstrating lack of obvious anatomical abnormality.

127x183mm (300 x 300 DPI)

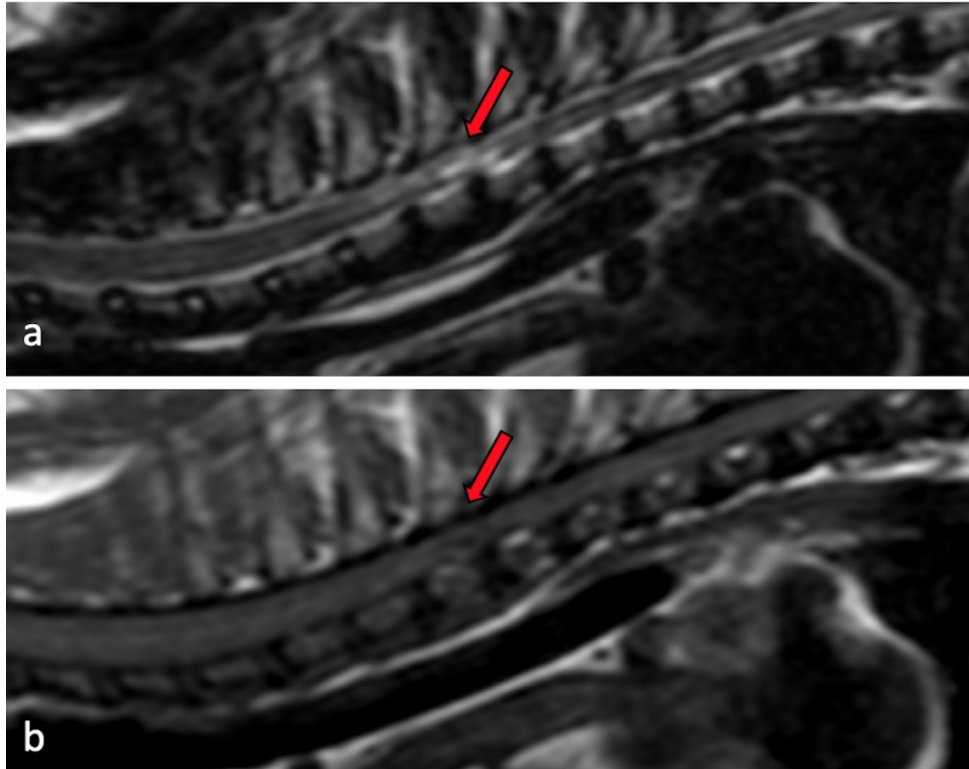


Figure 2 (a) T2W and (b) T1W sagittal images at the level of T4-T5 (red arrow) demonstrating a focal T2W hyperintense and T1W hypointense intraparenchymal lesion at the site of spinal cord compression. These imaging changes may be considered consistent with a cystic or cavitory type lesion.

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
 - WORSENERD

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: