CLINICAL FEATURES AND MAGNETIC RESONANCE IMAGING CHARACTERISTICS OF PRESUMPTIVE CONSTRICTIVE MYELOPATHY IN 27 PUGS

Filipa Lourinho[1], Andrew Holdsworth[1], J. Fraser McConnell[2], Rita Gonçalves[2], Rodrigo Gutierrez-Quintana[3], Carles Morales[4], Mark Lowrie[5], Raquel Trevail[1], Ines Carrera[6].

[1] Southern Counties Veterinary Specialists, Ringwood, UK;

[2] Dept. of Veterinary Science, Small Animal Teaching Hospital, University of Liverpool, UK;

[3] School of Veterinary Medicine, University of Glasgow, UK;

[4] Ars Veterinary Hospital, Barcelona, Spain;

[5] Dovecote Veterinary Hospital, Derby, UK;

[6] Radiology dept. Willows Referral Centre, Highlands Rd, Shirley, Solihull, UK.

Corresponding author:

Inés Carrera,ines.carrera.yanez@gmail.com

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ABSTRACT

Constrictive myelopathy has been described in pugs with paraparesis and is characterised by fibrous connective and granulation tissue within the dura mater causing spinal cord compression and focal gliosis. An association between constrictive myelopathy and caudal articular process (CAP) dysplasia is suspected; however, some studies have reported CAP dysplasia as an incidental finding. The imaging appearance of constrictive myelopathy is currently limited to a small number of cases. The aim of this retrospective, descriptive study was to detail the magnetic resonance imaging (MRI) characteristics and associated clinical signs of presumptive constrictive myelopathy in pugs.

Medical databases from five veterinary referral hospitals were reviewed to identify pugs with pelvic limb ataxia and paresis, that had a complete record of signalment, neurological examination, and MRI of the thoracolumbar spinal cord. The exclusion criteria were pugs with other conditions, such as unequivocal subarachnoid diverticula, hemivertebrae causing vertebral canal stenosis, intervertebral disc extrusions/protrusions, and multifocal/diffuse lesions.

27 pugs met the inclusion criteria. All cases were ambulatory with paraparesis and ataxia. Nearly 60% were incontinent. MRI revealed a focal myelopathy in all cases showing one or more of the following lesions: CAP dysplasia (25/27), focal subarachnoid space irregular margination (26/27) with circumferential or dorsal contrast enhancement (10/12), and a symmetric V-shaped ventral extradural lesion (23/27).

This study describes specific MRI features of pugs with presumptive constrictive myelopathy, which is hypothesised to be a consequence of chronic micro-motion. Our results may help in diagnosing and subsequently treating this condition, which may warrant vertebral stabilisation.

INTRODUCTION

Constrictive myelopathy has been previously described in pugs with paraparesis, and is characterised by the presence of fibrous connective tissue and granulation tissue affecting the dura mater that compresses the spinal cord and leads to focal gliosis.[1] The underlying pathophysiologic mechanism for constrictive myelopathy is not yet clear. However, a close association has been made between the development of this condition and the presence of an adjacent caudal articular process (CAP) dysplasia.[1] This type of vertebral malformation has been frequently identified in pugs[2], and is characterised by either the absence (aplasia) or incomplete formation (hypoplasia) of the CAP [3], with the reported prevalence varying between 64% up to 97% for the breed.[2,4] It is hypothesised that this malformation can create focal vertebral instability, which, over time, may result in peri-dural vascular changes and dural fibrosis or adhesions.[1] This may cause (1) cerebrospinal fluid (CSF) flow disturbances[1,5]; (2) hypertrophy of the *ligamentum flavum*[6]; (3) intervertebral disc (IVD) degeneration and ultimately (4) spinal cord edema and ischemia.[1,5,7] Clinically, this disease tends to have an insidious onset and manifest as a slowly progressive T3-L3 myelopathy,

76 characterised by paraparesis and pelvic limb ataxia that may or may not be accompanied by
77 urinary and/or faecal incontinence.[1,4,8]
78 The imaging appearance of constrictive myelopathy was first described in 2013 and was
79 limited to a small number of cases (n=11) in a single study.[1] The majority of these pugs
80 underwent computed tomography (CT) myelography, which revealed an abrupt attenuation,
81 narrowing or irregular course of the contrast columns from T11 to L1. Magnetic resonance
82 imaging (MRI) was performed in only four dogs, revealing right and left lateral narrowing of
83 the spinal cord, and indistinct articular processes. More recently, a different study
84 demonstrated that 90% of neurologically affected pugs had a vertebral malformation
85 immediately adjacent to a focal myelopathy.[9] However, the MRI description of the
86 myelopathy was limited to any focal compressive spinal cord lesion and/or focal
87 intramedullary T2-weighted (T2W) hyperintensity; no description of the extradural or
88 intradural lesion(s) responsible for the spinal cord compression was included. A recent
89 retrospective case series of pugs with thoracolumbar myelopathy and concurrent CAP
90 dysplasia recognised four different conditions based on the MRI findings: the most common
91 diagnosis was IVD protrusion, followed by subarachnoid diverticulae (SAD), pia-arachnoid
92 fibrosis, and vertebral instability.[10] The cases with pia-arachnoid fibrosis were likely
93 consistent with constrictive myelopathy, but were limited to a very small number of cases
94 (3/18).[10]
95 The aim of this retrospective study was therefore to provide a detailed description of the MRI
96 features of constrictive myelopathy in a larger number of pugs, and to correlate this with the
97 clinical signs.
98 The following hypotheses were made: 1. That CAP dysplasia in the caudal thoracic region
99 would be a common finding associated with focal myelopathy; 2. That the subarachnoid
100 space at the site of the focal myelopathy would be irregular in margination; 3. That post-

contrast images would reveal circumferential meningeal contrast enhancement; 4. That a bilateral ventrolateral extradural lesion (V-shaped) at the site of the focal myelopathy would be common; 5. That degenerative changes affecting the IVDs at the level of the CAP dysplasia would be a frequent finding; 6. That the spinal cord would show focal intramedullary T2W hyperintensity associated with the compressive lesions.

MATERIAL AND METHODS

This was a multi-center, retrospective, descriptive study, approved by The Research Ethics Committee of one of the institutions. Medical record databases from five referral institutions were reviewed retrospectively to identify pug dogs with paraparesis and pelvic limb ataxia consistent with a myelopathy between the T3 and L3 spinal cord segments. Dogs were included in the study if the performed MRI studies confirmed a focal T3-L3 myelopathy. Dogs were excluded if medical records or imaging studies were incomplete, if the diagnostic tests failed to identify a cause for the neurological signs, or if there was evidence of multifocal signs on both the clinical and MRI examinations. Additionally, dogs were excluded if the MRI studies revealed spinal cord compression caused by any of the following conditions: SAD (i.e. focal dorsal teardrop-shaped dilation of the subarachnoid space, often extending over two vertebral bodies, causing spinal cord compression, and intramedullary changes cranial or caudal to the lesion[11-13]); typical IVD extrusion (presence of ventral extradural material, often lateralised, consistent with nucleus pulposus, causing mass effect and compression/displacement of the subarachnoid and epidural spaces dorsally[14,15]); and/or protrusion (uniform and midline bulging of the annulus fibrosus and dorsal longitudinal ligament, also resulting in spinal cord compression and obliteration of the dorsal column[14,15]) and vertebral canal stenosis and kyphosis secondary to hemivertebrae.[16]

The signalment, neurological and MRI findings were recorded. The clinical signs were further categorised based on the onset (insidious versus acute), duration (chronic if more than one month), progression (progressive versus non-progressive), and the presence/absence of (1) spinal pain and (2) faecal and/or urinary incontinence.

All MRI examinations were assessed using a PACS workstation DICOM viewer (Osirix Imaging Software, version 3.9.2, Bernex, Switzerland). These studies were evaluated by three ECVDI-certified veterinary radiologists (IC, AH and FM) and an ECVN resident in training (FL), who were aware of the neurological deficits but were blinded to the remaining clinical history of each case at the time of the interpretation. If available, CT studies were reviewed to further evaluate the osseous structures. Histopathologic findings were also recorded when available.

The MRI abnormalities were evaluated for: (1) the presence of CAP dysplasia, subdivided into aplasia and hypoplasia. These changes were also defined as unilateral, bilateral, symmetric or asymmetric; (2) the appearance of the subarachnoid space (margination: regular or irregular; dimension: attenuated versus widened); (3) the presence of an extradural lesion (location, shape and signal intensity); (4) the presence of intramedullary lesions (location: spinal segment, grey and/or white matter; extent and signal intensity); (5) the degree of spinal cord compression, which was subjectively categorised as mild (if the spinal cord was reduced in size by <25% in transverse plane images when compared to non-affected spinal cord), moderate (25-50%), and severe (>50%); (6) the presence of IVD degenerative changes (solely at the site of the myelopathy, or widespread); (7) the presence of spondylosis at the site of the myelopathy; and finally (8) the presence of contrast enhancement in the studies where T1W post-contrast images were available (location: intramedullary, meningeal/dural; degree: mild, moderate, severe; pattern: linear, homogeneous, heterogeneous).

Statistical analyses were carried out by a veterinarian with a PhD and diplomate of the European College of Porcine Health and Management. Associations between the onset of the neurological signs and the clinical and MRI features were evaluated using Fisher's Exact tests or Wilcoxon-Mann-Whitney tests depending on the nature of the variables. All analyses were carried out using SAS 9.4 and the alpha level for determination of significance was set at 0.05.

RESULTS

Out of a total of sixty-one pugs with neurological signs consistent with a T3-L3 myelopathy, twenty-seven met the inclusion criteria. The remaining thirty-four were excluded based on the MRI findings: lack of a visible spinal cord lesion (n=2), poor quality study (n=1), classical, clearly defined subarachnoid diverticulae (n=15), simple IVD protrusion (n=5) or extrusion (n=2), hemivertebrae causing vertebral canal stenosis (n=5), and diffuse/multiple distant neuro-anatomical lesions (n=4) (e.g. multiple cervical IVD herniations causing significant spinal cord compression, diffuse syringomyelia).

Male dogs were overrepresented (n=18; 66.7%). The median age was 7.5 years (4.11-12). The majority of the dogs had an insidious onset of neurological signs (n=22), and only five had a sudden onset. In one case, acute and severe neurological signs were reported after the dog jumped from a bed two months prior to referral; the signs gradually improved and had remained static for several weeks before the dog was referred. Twenty-five dogs (92.6%) had a chronic duration (one month to two years), and only two dogs had a history of clinical signs for less than one month prior to the referral. Twenty-four dogs had a history of progressive clinical signs (88.9%), and only three dogs were reported to have no progression of the signs.

All dogs were ambulatory at the time of the referral but displayed pelvic limb ataxia and paraparesis. 14 dogs had lateralised signs (51.9%), characterised by proprioceptive deficits

that were more marked on one side. Segmental spinal reflexes were normal to increased in all cases. Eight dogs had faecal incontinence (29.6%), one had urinary incontinence (3.7%), and seven had both urinary and faecal incontinence (25.9%). The duration of the incontinence prior to the referral varied between a few weeks and several months, but this information was not available for all cases. A weak statistical correlation was found between the duration of the clinical signs prior to referral and the presence of incontinence ($P = 0.359$). Spinal pain was identified in five dogs (18.5%). No significant correlation was found between the onset of the clinical signs and the presence of spinal pain ($P = 0.252$).

Although MRI protocols and sequences varied between institutions, all MRI examinations were completed with dogs under general anaesthesia, using high-field-strength magnets: 1.5 Tesla (Hallmarq PetVet; Siemens Magnetom Essenza; Philips Ingenia Cx; Toshiba Vantage Elan) and a 1 Tesla MRI unit (Siemens Harmony). All dogs underwent 2D acquisition studies that included turbo spin echo T2W, and turbo spin echo T1-weighted (T1W) images. A group of these studies included short tau inversion recovery (STIR) images, T2* gradient recalled echo, steady state gradient echo (B-FFE), T1W post-contrast images and T1W post-contrast with fat saturation (mDIXON) (0.1mmol/kg intravenous gadopentetate dimeglumine). The sequence's parameters are summarised in table 1. Consensual evaluation of the MRI studies led to complete agreement between readers. The findings are available in tablet 2. At the site of the myelopathy, 92.6% of the dogs (n=25) had concomitant CAP dysplasia (figure 1). Of these, 21 had CAP dysplasia affecting multiple adjacent vertebrae (84%), and four affecting only one vertebra (16.7%). At the site of the myelopathy, the articular process joints were bilaterally aplastic in 16 cases (64%) and bilaterally hypoplastic in 9 cases (33.3%), with some degree of asymmetry noted in 7 cases (28%). CT was available in 9 dogs (33%), which confirmed the MRI findings in all but one case. In this case, CT images revealed that the CAPs were hypoplastic whilst on MRI they appeared hyperplastic, likely due to soft tissue

proliferation surrounding the articular processes. Of the two cases with normal CAPs at the site of the myelopathy on MRI, only one had a CT to confirm this. The myelopathy sites were predominantly caudal to T10, with the exception of one case which was at T8-9. In this case, multiple adjacent sites of CAP dysplasia were identified in the available acquired transverse images (from T6-7 to T9-10), which did not include the vertebral column caudal to T10.

The subarachnoid space had an abnormal appearance on MRI in all of the cases. The subarachnoid space had a focal, irregular and stellate shape in 26 dogs (96.3%), predominantly in the dorsal aspect. The T2W images were characterised by a mixture of increased signal (which corresponds to CSF) with abnormal hypointense material/bands within the subarachnoid space, which was homogeneously hypointense in the T1W images (figure 2). The subarachnoid space was irregularly marginated and mildly widened in 7 cases, partially attenuated in 13 cases, and had regions of widening and attenuation in 3 cases. In one case, there was only circumferential attenuation of the subarachnoid space without any obvious irregularity. Post-contrast images were available in 12 cases (44%). Of these, 2 had no contrast enhancement (17%), 5 had circumferential enhancement of the meninges (42%) (figure 3A-C), and 5 had a focal enhancement of the meninges dorsally (42%) (figure 3D-F).

24/27 dogs (88.9%) had an extradural lesion at the site of the myelopathy. 23 dogs had a solitary lesion at the level of the IVD space. In one dog, two adjacent spaces were affected, resulting in a total of 25 extradural lesions. Of these, 24 had a bilateral and ventrolateral distribution (with a V-shaped appearance), and were hypointense to isointense (to normal spinal cord) on T1W images and homogeneously hypointense on T2W images (figure 2, arrow-heads). The majority (n=22) were caudal to T10 (four at T10-11, seven at T11-12, seven at T12-13 and four at T13-L1), and the remaining 3 were at the T8-9 IVD space (of which two did not have adjacent CAP dysplasia). Three dogs did not have an extradural lesion at the myelopathy site.

IVD changes (characterised by a reduction in T2W signal intensity, and mild or no reduction of the IVD volume), were confined to the site of the myelopathy in 6 cases (22.2%) and widespread in 20 cases (74.1%). Only one case did not have any gross IVD changes. Spondylosis was identified at the myelopathy site in 8 cases (29.6%). All cases had focal spinal cord compression at the level of the extradural lesion(s) and/or where the subarachnoid space was abnormal. The degree of spinal cord compression was mild in 7 cases (25.9%), moderate in 14 cases (51.9%), and severe in 6 cases (22.2%).

All cases had a focal intramedullary lesion at the level of the spinal cord compression (figure 2, dotted arrows), although no statistically significant association was found between the extent of these changes and the degree of spinal cord compression (P = 0.605). All intramedullary lesions were T2W hyperintense compared to normal spinal cord, T1W isointense, and did not display contrast enhancement. In the majority of cases, the changes extended over one to two vertebral body lengths (n=13; 48.1%), or over less than one vertebral body length (n=12; 44.4%). The changes extended over more than three vertebral body lengths in 2 cases only (7.4%). These changes affected both grey and white matter, although the grey matter seemed to be affected more consistently, and were mostly located dorsally. No significant correlation was found between the duration of the clinical signs and the extension of the intramedullary changes (P = 0.369). In the two cases with normal CAPs, the intramedullary lesion was located at T8-T9 in one case, and centred over the mid-body of T8 in the other case. Both had a T8-9 extradural lesion, accompanied by a focal irregularly marginated subarachnoid space appearance and concomitant spondylosis.

Histopathology from the extradural lesion was only available in one case, which demonstrated dense collagen bundles interspersed with attenuated fibroblasts that were arranged in parallel streams. Scattered clusters of mineralized matrix and occasional small areas of cartilaginous metaplasia were present within the collagenous matrix.

Overall, of all the cases with CAP dysplasia adjacent to the myelopathy site (25/27), all showed intramedullary changes, 88% had an extradural lesion (22/25), 96% also had an irregular subarachnoid space (24/25). The combination of these three MRI features was found in 81.5% of the cases with CAP dysplasia.

DISCUSSION

This study clarified a distinctive MRI appearance of focal thoracolumbar myelopathy in pugs with pelvic limb proprioceptive ataxia and paresis. In 74.1% of the cases, this was characterised by a combination of CAP dysplasia, irregular subarachnoid space, bilateral ventrolateral extradural lesion, moderate focal spinal cord compression, and intramedullary changes. These imaging features, in combination with the signalment and neurological findings, are highly suggestive of a focal constrictive myelopathy.

CAP dysplasia has been suggested to play an important role in the development of constrictive myelopathy in pugs[1] and this was supported by the present study, where 92.6% of the cases had CAP dysplasia adjacent to the myelopathy. When compared to other "screw-tailed" breeds, pugs seem to have a higher number of affected vertebrae per individual, often involving multiple adjacent vertebrae, and predominantly in the caudal thoracic compartment. (i.e. caudal to T10).[2,17] This corresponds to a transitional portion of the spine, which makes it more susceptible to instability in the presence of CAP dysplasia.[3] The articular process joints are believed to contribute up to 30% of the stability of the vertebral column, preventing spinal extension and axial rotation in the caudal thoracic region, whereas cranially they play a more important role in weight bearing rather than in restricting motion.[3,18] The high number of cases with a thoracolumbar myelopathy associated with CAP dysplasia found in this study (96% caudal to T10) supports the hypothesis that micro-instability may be one of the main causes for the development of adjacent myelopathies in

this breed. Although CAP malformation has been identified in unaffected dogs[2,19], this theory has been suggested in multiple studies.[1,2,10,17,20] Interestingly, whilst this osseous abnormality is not limited to pugs[2,3], to the author's knowledge this particular myelopathy has never been reported in any other breed. It is also interesting to note that pugs with hemivertebrae were recently reported to be 10 times more likely to become clinically symptomatic when compared to other brachycephalic breeds with the same malformation, and a clear cause for this discrepancy has not yet been determined.[21] This raises concern that other factors are likely to contribute to the development of neurological signs secondary to vertebral malformations in this breed, either by exacerbating the assumed micro-instability (e.g. abnormal posture, weaker trunk muscles, etc.) or through an alternative unknown mechanism. More studies are necessary to investigate these hypotheses.

In this study, the identification of CAP dysplasia was assessed on MRI in all cases. The authors believe that images acquired by high-field MRI scanners (with good quality, appropriate slice thickness (thin slices) and/or 2D GE sequences) are sensitive enough to identify this vertebral malformation, which was supported by the fact that, when available, CT studies confirmed the MRI findings in all cases but one. CT in addition to MRI can still be useful, especially when the CAPs appear normal or hyperplastic on MRI, when the image quality is suboptimal, or when additional information is required for surgical planning. Only two cases in this study did not show evidence of CAP dysplasia. In these cases, the reason for the development of the myelopathy (which had a similar MR appearance to the cases with CAP dysplasia) remains unknown. More dogs would be required to study this in more detail.

A strong correlation between the diagnosis of SAD and the presence of an adjacent CAP dysplasia in pugs has been found.[10,22] Interestingly, histopathology of the affected dura mater revealed fibrosis in some cases[22], which is similar to the results encountered in dogs diagnosed with constrictive myelopathy.[1] The widened subarachnoid space resulting from the

dural adhesions can sometimes be mistaken for SAD, making a clear differentiation between these two entities challenging.[10] Classical SAD has been described as a uniform teardrop-shaped lesion adjacent to the spinal cord and contiguous with the subarachnoid space, typically with a dorsal location and without contrast enhancement.[23] This study allowed the recognition of MRI features that may help to distinguish SAD from the dural fibrosis/adhesions that lead to constrictive myelopathy. These comprise the subarachnoid space irregularity (likely to be the result of chronic irritation and subsequent focal cicatrix formation affecting the meninges and consequently the CSF flow[12,23-25]) with mixed T2W signal intensity due to the hypointense material/bands within the subarachnoid space, which can subsequently cause either a widening or an attenuation of this space, or a combination of both. In addition, the post-contrast images showed a circumferential or focal dorsal enhancement of the meninges in the majority of the cases. We suggest that post-contrast studies in cases of suspected focal constrictive myelopathy may be valuable to identify focal fibrosis. Attempts to differentiate these two conditions have also been made in humans.[26] Abnormalities of the septum posticum (a membrane connecting the pia to the arachnoid membrane) have been hypothesised to be the origin of cyst-like intradural lesions (i.e. lesions where there is communication of CSF spaces around membranes as opposed to a true compartmentalization) that lead to spinal cord compression.[26] Little is known about the relevance of this structure in dogs, and therefore a direct extrapolation cannot be made.

One of the MRI features encountered in the majority of the cases included in this study (85.2%) was a characteristic V-like shaped extradural lesion. Histopathological analysis available from one of these lesions was consistent with fibrotic material and chondroid metaplasia. The reason for this characteristic MR appearance is unknown, although it may correspond to the “right and left lateral narrowing of the spinal cord” described by the authors who first reported this condition.[1] Other similar bilobed-shaped lesions have been associated

to the presence of the recently described meningovertebral ligament, which causes an anatomic boundary that leads to this characteristic shape, regardless the underlying pathologic process.[27] Extradural lesions have not been reported in association with constrictive myelopathy. However, given the high number of affected cases in this study, we suspect this may occur concomitantly, either as part of the condition or as another consequence of the presumed focal micro-instability. More samples would be required, including analysis from post-mortem specimens, to accurately determine the nature of these imaging findings. The absence of an extradural lesion in some cases (n=3) may be suggestive of either an earlier stage of this disease, or alternatively a different manifestation of this disease where IVD protrusion does not develop. Spinal cord trauma/contusion associated with the presumptive dynamic factor suspected with this condition could also explain the development of a focal myelopathy without an adjacent extradural lesion. However, no strong correlation was found between the onset and the presence or absence of an extradural lesion ($P = 0.999$). It is also interesting to note that only a few cases had spondylosis at the affected site (8/27) and that the majority of the dogs had widespread degenerative IVD changes rather than focal IVD changes. In humans it has been demonstrated that destruction of the articular process joints accelerates degeneration of the adjacent IVD, due to the transference of axial loads to the annulus and anterior longitudinal ligament.[28] This was not encountered in our study. Nevertheless, pugs are a chondrodystrophic breed and therefore these results may be simply the reflection of early degenerative IVD changes, rather than a result of an abnormal load on the vertebral column itself.

All cases had some degree of spinal cord compression, predominantly ventral and dorsal, caused by the ventral extradural lesion and the irregularly marginated dorsal subarachnoid space respectively. This chronic compression, combined with the suspected micro-motion of the adjacent vertebra, is believed to gradually aggravate the myelopathy, which in all but one

case was represented by intramedullary changes at the level of the compression. However, no correlation was found between the duration of the clinical signs and the extent of these intramedullary changes, as it did not exceed two vertebral body lengths in the majority of the cases (92.5%), and it was located focally over the IVD space in nearly half of the cases. This differs from the intramedullary changes reported with SAD, which usually extend cranially or caudally to the point of maximal spinal cord compression[13], rather than being focal at the level of the compression as described in this study. These changes are likely to represent a combination of neuronal necrosis, apoptosis and gliosis, as are often seen in chronic spinal cord lesions[1,22,29], and accompanied by syrinx or pre-syrinx formation.[10,22]

The median age of dogs with constrictive myelopathy was previously reported as 7.7 years.[1] This is in agreement with this study (median 7.5 years; range 4.11-12 years) and it can be explained by the chronic nature of the lesions, which may ultimately only start to manifest neurological signs later in life.

The vast majority of the dogs in this study had an insidious onset of signs, which were chronic and progressive. However, interestingly, four cases developed signs suddenly. This information was provided by the owners and therefore its accuracy cannot be certain. It is possible that subtle neurological signs were present before they became noticeable to the owner, or that high impact activity could have caused an acute onset of neurological signs, unmasking a condition that was already subtly developing.

Loss of continence was a frequent feature in this study (over 60% of the cases). This is believed to be secondary to a lesion in the dorsal portion of the spinal cord that interferes with the cranial projecting sensory pathway for both defecation and urination.[30] Indeed, spinal cord histopathology of constrictive myelopathy was previously reported to show a marked dorsolateral flattening of the spinal cord with significant tissue loss.[1] Incontinence has also been reported in other spinal cord disorders[12,29,31], such as SAD.[12] Based on this, the

relationship between incontinence and the presence of a widened subarachnoid space was evaluated, but no statically significant correlation was seen (P = 0.999). It also remains unclear why some dogs develop faecal incontinence only, and others urinary incontinence or both, suggesting that a different pathway for micturition and defecation must exist.[31] In agreement with previous studies[1,10], spinal hyperaesthesia was very uncommon (15.4%) and no significant correlation was found between this and the onset of the neurological signs (P = 0.252).

Limitations of our study include its multi-institutional and retrospective nature, and the inherent variability in the available clinical information, as well as variability in the MRI sequences that were performed. The retrospective and clinical descriptive nature of the study prevented histopathology/post-mortem evaluation of the MRI findings to confirm the diagnosis of constrictive myelopathy in all cases, which is considered one of the main limitations of this study. Spinal cord conditions that have already been well characterised in the current literature were excluded from this study. However, it is not possible to say whether some of these conditions were a different manifestation of the same disorder, such as the development of SAD. The widened subarachnoid space seen in some cases can sometimes resemble SAD and it may, in fact, be a variant of this disease in this breed. Post-contrast T1W transverse images, as suggested in this study, may help distinguishing this from the classical SAD, but follow-up MRI studies could provide more information regarding a potential correlation between these two entities. Detailed evaluation of osseous structures and the three-dimensional trajectory of the vertebral column in all planes is best achieved by CT[16], which was not available for every case in this study. This limitation was tackled by the fact that all images were reviewed by three board-certified radiologists who felt confident that they could identify CAP dysplasia from the MR images. Nevertheless, the authors

encourage the readers to combine other imaging modalities for evaluation of osseous structures when this is deemed suboptimal with MRI alone, and/or for surgical planning.

In conclusion, this study describes characteristic MRI features of pugs with presumptive constrictive myelopathy, which appears to be a multifactorial disorder occurring as a consequence of suspected chronic micro-motion. Our results may help (1) recognizing this condition using MRI alone, (2) alert for the potential necessity of combining different imaging modalities to evaluate the osseous structures, and (3) sets the foundation for further studies to assess treatment options, as it may warrant vertebral stabilisation.

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Author Contributions:

Category 1

(a) Conception and Design : Inés Carrera

(b) Acquisition of Data : Filipa Lourinho, Andrew Holdsworth, J. Fraser McConnell, Rita Gonçalves, Rodrigo Gutierrez-Quintana, Carles Morales, Mark Lowrie, Inés Carrera.

(c) Analysis and Interpretation of Data : Filipa Lourinho, Andrew Holdsworth, J. Fraser McConnell, Raquel Trevail, Inés Carrera.

Category 2

(a) Drafting: Filipa Lourinho, Inés Carrera

(b) Intellectual Content : Filipa Lourinho, Andrew Holdsworth, J. Fraser McConnell, Rita Gonçalves, Rodrigo Gutierrez-Quintana, Carles Morales, Mark Lowrie, Raquel Trevail, Ines Carrera.

Category 3

(a) Final Approval: Filipa Lourinho, Andrew Holdsworth, J. Fraser McConnell, Rita Gonçalves, Rodrigo Gutierrez-Quintana, Carles Morales, Mark Lowrie, Raquel Trevail, Ines Carrera.

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LEGENDS

FIGURE 1 T2W parasagittal (A), T1W transverse (B and C) MR images and CT (D, E and F) of the thoracolumbar vertebral column of a pug. The parasagittal planes demonstrate the difference between the anatomically intact (arrow heads) and aplastic (arrows) CAP on both MRI (A) and CT (D), which can also be appreciated in the transverse planes (B, C, E and F), where the CAPs are absent on B and E (arrows) and present on C and F (arrows).

FIGURE 2 T2W MR images of three different cases with presumptive constrictive myelopathy. The sagittal planes (images A, E and H) show a focal ill-defined intramedullary T2W hyperintensity (dotted arrows) in all cases. The transverse images correspond to the dashed line identified by the same letter on the sagittal images. In all cases, the subarachnoid space is irregularly marginated and uneven in width immediately cranial (images B1, F1, I1 and J1, see arrows) and/or caudal (figure D1) to the extradural lesion. The bilobed appearance of the extradural lesion is highlighted by arrowheads on images C1, G2 and K1.

FIGURE 3 MR T1W images, pre (A and D) and post gadolinium administration (B and E) showing circumferential (B) and dorsal (E) contrast enhancement of the meninges. The contrast uptake can be better identified on the subtraction images (C and F).

TABLE 1 This table summarizes the different high-field-strength magnets used and the corresponding image acquisition parameters for each of them (slice thickness, repetition time (TR), echo time (TE), inversion recovery (IR), echo train length (ETL), number of signals averaged (NSA) and flip angle for gradient sequences).

TABLE 2 Table summarizing the number and percentage of cases with different combinations of MRI features (i.e. adjacent CAP dysplasia, irregular subarachnoid space, extradural lesion, intramedullary T2W hyperintensity, post-contrast images, degree of spinal cord compression and localisation). The symbol "✓" means the feature was present, and "✗" absent.