PAPER

Clinical and magnetic resonance imaging features of canine compressive cervical myelopathy with suspected hydrated nucleus pulposus extrusion

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Objective: To describe clinical and magnetic resonance imaging features in dogs with compressive cervical myelopathy due to acute suspected hydrated nucleus pulposus extrusion.

Design: Retrospective case series.

Animals: Ten dogs with compressive cervical myelopathy caused by acute suspected hydrated nucleus pulposus extrusion.

Materials and Methods: Medical records and magnetic resonance images of dogs evaluated from 2005 through 2010 were reviewed. The inclusion criteria were clinical signs compatible with cervical myelopathy, magnetic resonance imaging of the spine performed within 48 hours after onset, magnetic resonance imaging findings consistent with compressive suspected hydrated nucleus pulposus extrusion, complete medical records and follow-up information.

Results: Six dogs were presented with acute onset tetraplegia and four dogs with acute onset of non-ambulatory tetraparesis. Compromised respiratory function was present in three dogs. Compressive suspected hydrated nucleus pulposus extrusion was found on magnetic resonance imaging at the C4-C5 (n=6), C3-C4 (n=3) and C5-C6 (n=1) intervertebral disc spaces. Seven dogs underwent surgical treatment and three dogs were treated conservatively. All dogs except one regained ambulatory status within two weeks after the onset, and had a favourable outcome.

Clinical Significance: Compressive myelopathy caused by acute suspected hydrated nucleus pulposus extrusion has not been reported previously and, even though neurological deficits can be severe on presentation, outcome is favourable.

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INTRODUCTION

Cervical disc herniation has been reported in 21 to 44% of dogs with intervertebral disc (IVD) disease (Fluehmann and others 2006, Itoh and others 2008). Hansen (1951) described two types of IVD degeneration classified as Hansen type I or chondroid degeneration and Hansen type II or fibroid degeneration, which can result in IVD herniation and spinal cord compression. Both types of IVD degeneration occur in the cervical region but in the dog Hansen type I is reported to be more common (Cherrone and others 2004).

Griffiths (1970) described another type of intervertebral disc herniation, which may also occur in the cervical spine (De Risio and others 2009). This type of extrusion occurs when

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non-degenerated nucleus pulposus extrudes during strenuous exercise or trauma causing spinal cord contusion with little or no residual spinal cord compression. Several terms have been used to describe this condition including acute, non-compressive nucleous pulposus extrusion (De Risio and others 2009), high velocity, low-volume disc extrusion (Lu and others 2002) or traumatic disc extrusion (Chang and others 2007).

This study was prompted by a clinical impression that acute, compressive hydrated nucleus pulposus extrusion (HNPE) exists in dogs. The purpose was to describe the neurological signs, magnetic resonance imaging (MRI) findings, treatment and outcome in dogs with compressive cervical myelopathy due to acute suspected HNPE.

MATERIALS AND METHODS

Medical records and magnetic resonance (MR) images of dogs presented to the Animal Health Trust from January 2005 to October 2010 were reviewed. The inclusion criteria were clinical signs compatible with cervical myelopathy, MRI of the cervical spine performed with a 1.5-Tesla (high field) unit within 48 hours after the onset of neurological signs, MRI findings consistent with compressive suspected HNPE, complete medical records and follow-up information.

Information retrieved from the medical records included age, sex, breed, bodyweight, body condition score, type of onset (acute <24 hours, subacute 24 to 48 hours or chronic >48 hours), type of physical activity at the onset of clinical signs, medication administered by the referring veterinarian, physical and neurological examinations at presentation, neurolocalisation, MRI findings, type of treatment (surgical or conservative), neurological/clinical status at discharge, recheck examination and telephone follow-up at least four months after discharge.

Body condition score was assessed using a scale of one to nine, with one being very underweight and nine being very overweight. A body condition score of five is considered ideal (Laflamme 1997). Dogs were classified as chondrodystrophoid or non-chondrodystrophoid type (Hansen 1951, Braund and others 1975, Martinez and others 2000, Martinez and others 2007).

Neurological dysfunction was classified as tetraplegia with nociception, non-ambulatory tetraparesis and ambulatory tetraparesis with or without ataxia. Dogs were considered ambulatory if they could walk without support. The presence of spinal hyperaesthesia was also recorded.

A 1.5-Tesla magnet (GE Signa, GE Medical System) was used for all dogs. T2-weighted (T2W) fast spin-echo (FSE) images were obtained in the dorsal and sagittal plane in all the dogs from C1 to T2 vertebral bodies. Transverse plane T2W FSE images of the affected IVD space(s) were obtained in all the dogs. When available, T1-weighted (T1W) FSE sequences before and after intravenous bolus administration of gadopentetate dimeglumine at 0·1 mL/kg of bodyweight (Multi-Hance®, Bracco Imaging SpA or Gadovist®, Bayer Schering Pharma) and T2* gradient echo (GE) were also reviewed. All MR images were reviewed by two board-certified radiologists to obtain a consensus on the diagnosis of compressive suspected HNPE. Magnetic resonance imaging findings consistent with compressive suspected HNPE were defined as extradural compressive material immediately above an IVD space, which was isointense to hydrated nucleus pulposus, ill-defined dorsal annulus, with reduction in nuclear volume and narrowing of the affected IVD space. The remaining material in the intervertebral disc space is also isointense to hydrated nucleus pulposus. The degree of spinal cord compression (SCC) was calculated on transverse T2W images as previously described (Ryan and others 2008). The cross-sectional area (CSA) of the spinal cord at the point of maximum compression was measured by tracing the outline of the cord using a commercial software (Osirix version 3.8.1 DICOM viewer; Fig 1a). Using the same method, the region of normal spinal cord closest to the site of the compression was identified and the CSA of the non-compressed spinal cord was also measured (Fig 1b). Magnetic resonance imaging findings consistent with normal spinal cord on transverse T2W images were defined as non-compressed spinal cord surrounded by a well-demarcated high-signal-intensity area representing cerebrospinal fluid and

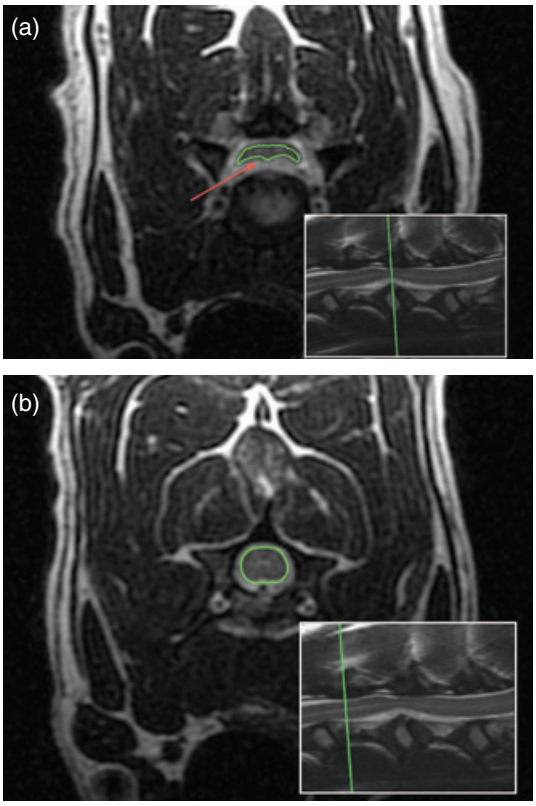

FIG 1. (a) Transverse T2-weighted image with the outlined cross-sectional area of the spinal cord at the level of maximal dorsoventral compression. The suspected extruded material (red arrow) lies symmetrically on the midline ventral to the spinal cord. The image below right shows the cross reference of the transverse view. (b) Transverse T2-weighted image with the outlined cross-sectional area of the spinal cord at the region of the normal spinal cord closest to the site of compression. The image below right shows the cross reference of the transverse view

epidural fat. The degree of SCC, expressed as a percentage, was calculated by the formula

$$\frac{\text{CSA of normal cord} - \text{CSA of cord under maximal compression}}{\text{CSA of normal cord}} \times 100$$

Conservative management consisted of strict, confined rest for four to six weeks, anti-inflammatory medication, muscle relaxants (when needed), analgesics, physiotherapy and the use of a harness rather than a collar. Surgical treatment consisted of a decompression procedure via ventral slot. Surgical decompression was always followed by postoperative care, as described for the conservative treatment.

Reassessment was performed between four to six following discharge. The owners of all dogs which were still alive at the end of the study were contacted by telephone to determine the long-term outcome.

RESULTS

Ten dogs met the inclusion criteria (Table 1). The median age at diagnosis was 9 years (range 8 to 13 years). The median bodyweight was 18 kg (range 10 to 37 kg). The median body condition score was 5 (range 4 to 6). Seven dogs were classified as non-chondrodystrophoid and three dogs as chondrodystrophoid types. All dogs had an acute onset of cervical myelopathy and progressive deterioration within 24 to 48 hours from the onset. In all but one of the dogs the onset of the clinical signs was not associated with any type of physical activity (running, jumping or playing) or traumatic event (road traffic accident). In the remaining dog the onset of clinical signs appeared whilst running, without any witnessed traumatic event.

Before the neurological assessment, two dogs received one dose of non-steroidal anti-inflammatory medication (meloxicam 0·2 mg/kg, Metacam, Boehringer Ingelheim), one dog was on long-term medication due to skin allergy 0·35 mg/kg prednisolone (Prednidale, Dechra) once a day, and seven dogs had not received any medication.

Physical examination at presentation was unremarkable in eight dogs. Two dogs showed respiratory dysfunction; both were recumbent, cyanotic, had abdominal breathing effort and were immediately treated with oxygen supplementation.

Neurological examination at presentation showed tetraplegia with nociception in six dogs, and non-ambulatory tetraparesis in four dogs. The neurolocalisation was to the C1-C5 spinal cord segments in three dogs and to the C6-T2 spinal cord segments in six dogs. The remaining dog had an acute onset of lower motor neuron signs in all four limbs (decreased to absent withdrawal reflexes in all four limbs, and decreased patellar reflexes in both pelvic limbs). Within less than 4 hours the neurolocalisation changed to C1-C5 spinal cord segments, which supported the presumptive diagnosis of spinal shock in this dog. Cervical discomfort was present in two dogs and absent in eight dogs.

T2-weighted images were obtained in all dogs, T1W images in eight dogs (seven with intravenous contrast administration) and T2* GE images in six dogs. The affected IVD was C4-C5 in six dogs, C3-C4 in three dogs and C5-C6 in one dog. Two dogs with the neurolocalisation of C6-T2 spinal cord segments had the compressive suspected HNPE at C3-C4 IVD space. In all the dogs, the suspected extruded IVD material was located ventral to the spinal cord, in the midline and centred immediately

Table 1. Details of signalment, clinical presentation, site of intervertebral disc (IVD) herniation, spinal cord compression (SCC) treatment and outcome in dogs with suspected hydrated nucleus pulposus extrusion

Dog	Breed	Age (years)	Gender	Bodyweight (kg)	Onset (hours)	Clinical signs at presentation	Site of the IVD herniation	SCC (%)	Treatment	Regain ambulation (days)
1	Labrador	9	Mn	37	<24	Non-ambulatory tetraparesis	C4–C5	29	Ventral slot	5
2	Labrador	9	Mn	30	<24	Tetraplegia with nociception	C4–C5	51	Ventral slot	Died due to a cardio-respiratory arrest
3	Labrador	9	Me	35	<24	Tetraplegia with nociception	C4–C5	32	Ventral slot	1
4	Working sheepdog	11	Me	21	<24	Non-ambulatory tetraparesis	C3–C4	11	Conservative	1
5	English springer spaniel	9	Mn	19	<24	Non-ambulatory tetraparesis	C4–C5	10	Conservative	1
6	Jack Russel terrier cross	13	Fn	11	<24	Tetraplegia with nociception	C3–C4	33	Ventral slot	12
7	Basenji	9	Me	10	<24	Tetraplegia with compromised respiration	C3–C4	50	Ventral slot	8
8	Swedish vallhund	12	Fn	13	<24	Tetraplegia with nociception	C4–C5	25	Ventral slot	9
9	Lhasa apso	12	Me	11	<24	Non-ambulatory tetraparesis	C5–C6	14	Conservative	2
10	Border collie cross	8	Mn	16	<24	Tetraplegia with spinal shock and compromised respiration	C4–C5	48	Ventral slot	14

Mn Male neutered, Me Male entire, Fn Female neutered

dorsal to a slightly narrowed IVD space. On sagittal T2W images the suspected extruded IVD material was difficult to distinguish from epidural fat and cerebrospinal fluid because of its similar signal intensity, but spinal cord elevation and compression over the affected disc space was clearly shown (Fig 2). Eight dogs had a characteristic "seagull sign" appearance of the suspected hydrated, extruded IVD material on transverse T2W images (Fig 3). The median SCC was 31% (range 10 to 51%). Eight dogs had focal intramedullary high signal intensity on T2W images immediately above the suspected nucleus pulposus extrusion. This focal area was isointense on T1W images, non-enhancing on post-contrast T1W images and isointense to high signal intensity on T2* GE and was thought likely to represent spinal cord contusion. Mild, diffuse contrast enhancement of the suspected extruded IVD material was present in three of the six dogs in which contrast-enhanced images were acquired.

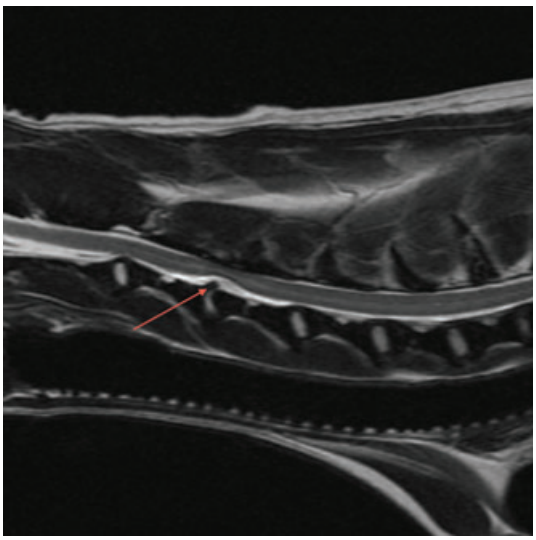

FIG 2. Sagittal T2-weigthed image showing suspected extradural compressive material above C3–C4 extending over cranial C4 and isointense to hydrated nucleus pulposus. Ill-defined dorsal annulus (red arrow), reduction in the nuclear volume and narrowing of the affected IVD space are also evident

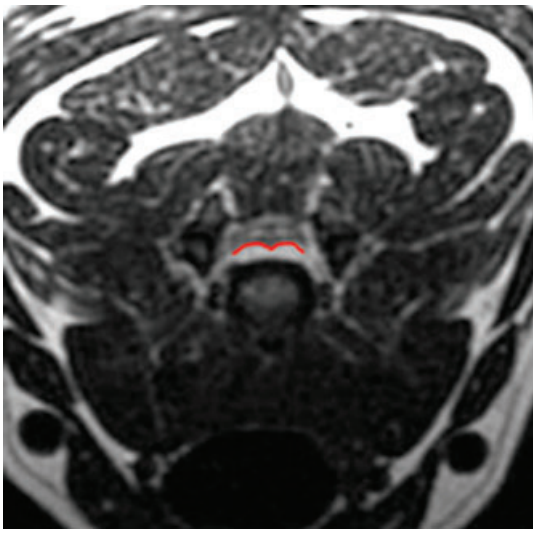

FIG 3. Transverse T2-weighted image of a suspected hydrated nucleus pulposus extrusion. The extruded material lies symmetrically on the midline ventral to the spinal cord. The margin of the extruded material resembles a "seagull"

Surgery was performed in seven dogs, based on the degree of SCC and the severity of the clinical presentation. All dogs that underwent surgery had a SCC more than 24%. A ventral slot was performed in all seven dogs and the extruded material removed from the epidural space was white, opaque and gelatinous in appearance. One dog died on recovery after the surgical procedure due to respiratory dysfunction followed by cardiac arrest. This dog had SCC of 51% with severe, focal, intramedullary hyperintensity on T2W FSE images and the spinal cord visibly appeared soft and discoloured at surgery, suggesting malacia. Three dogs had less severe SCC and were managed conservatively.

All dogs that survived regained ambulatory status. The median time for dogs that underwent surgery to regain ambulatory status was five days (range one to 14 days). Dogs that were managed conservatively regained ambulatory status with a median time of one day (range one to two days).

Nine dogs had neurological reassessment at four to six weeks after the onset of the clinical signs and all had remained ambulatory and comfortable.

All owners were contacted subsequently via telephone. Seven dogs were still alive, comfortable and ambulatory with a median follow-up of 352 days (range 159 to 1158 days). One dog had died due to an unrelated condition (diabetes mellitus) 12 months after regaining ambulatory status. The remaining dog had died due to an unknown cause five years after regaining ambulatory status.

DISCUSSION

Cervical myelopathy caused by acute, compressive, suspected HNPE as described herein, has not been previously reported in dogs. This study reports signalment, clinical signs, MRI findings, treatment and outcome in 10 dogs with this condition.

The affected dogs were both non-chondrodystrophic and chondrodystrophic. The median age at presentation was nine years, and from the results of this study it is therefore unlikely that acute, cervical, compressive, suspected HNPE affects only young dogs.

Cervical hyperaesthesia can occur from disease on any of the different structures surrounding the vertebral column, including nerve roots, meninges, the annulus of the intervertebral disc, vertebral periosteum, joint capsules, the epaxial musculature and the ligaments (Platt 2004). Cervical hyperaesthesia is the most common clinical sign in acute onset cervical disc herniation in dogs (Sharp and Wheeler 2005). Non-ambulatory tetraparesis is an infrequent manifestation of cervical disc herniation in dogs and tetraplegia with loss of nociception is not recognised because respiratory arrest occurs with such a lesion (Cherrone and others 2004, Levine and others 2007, Hillman and others 2009).

The majority of the dogs affected by acute, compressive suspected HNPE did not have cervical discomfort at presentation and all the dogs were non-ambulatory. This study shows that dogs with acute, compressive suspected HNPE present with severe neurological deficits but that cervical hyperaesthesia is not a common clinical sign. Three of the dogs which did not present with cervical discomfort had received anti-inflammatory medication before being referred, which may have influenced the clinical status. In humans, lateral disc herniations are common causes of radiculopathy and radicular pain, whereas midline disc herniations mainly cause myelopathy (Lestini and Wiesel 1989, Dubuisson and others 1993). All the dogs in this study had a midline suspected HNPE without nerve root involvement, perhaps explaining why cervical hyperaesthesia is not the main feature in this type of extrusion. Similar clinical presentation could occur with ischaemic myelopathy (De Risio and others 2009, De Risio and Platt 2010) or traumatic intervertebral disc extrusion (Chang and others 2007, De Risio and others 2009). Ischaemic myelopathy is characterised by peracute onset of non-progressive and non-painful myelopathy but this myelopathy is predominantly lateralised due to the asymmetric distribution of the central artery at different spinal cord segments (De Risio and Platt 2010), which was not a clinical feature on the present study. Traumatic intervertebral disc extrusion is also characterised by an acute, also often asymmetric and non-progressive myelopathy (Griffiths 1970). This type of intervertebral disc extrusion typically occurs after trauma or intense exercise, which was not a feature in the present study. Differentiating between these two conditions may depend on presentation, clinical assessment and high quality, high resolution MRI images.

Respiratory complications are the most common cause of morbidity and mortality in humans with acute cervical spinal cord injury, with an incidence of 36 to 83% (Berlly and Shem 2007). Approximately 5% of dogs with cervical spinal cord disorders may need ventilatory support perioperatively (Beal and others 2001). The respiratory system can be compromised by cervical spinal cord disorders mainly due to a disruption of the reticulospinal tracts, phrenic nuclei or phrenic nerve, which supplies the diaphragm (King 1987, Evans and Kitchell 1993). In this study three dogs had severe respiratory dysfunction, two of which were affected presurgically and one postsurgically. All three dogs were tetraplegic and had severe spinal cord compression. Respiratory function should be assessed and monitored thoroughly in all dogs that present with severe neurological dysfunction due to a cervical spinal cord disorder as the outcome may be compromised if appropriate respiratory support is not provided.

In this study, one dog had transient decreased segmental spinal reflexes in all four limbs. This phenomenon has been previously described in dogs with acute thoracolumbar spinal cord injury (Smith and Jeffery 2005, de Lahunta and Glass 2009, De Risio and others 2009) but has not been described in dogs with acute cervical spinal cord injury. This phenomenon is known as spinal shock, suggested to be associated with sudden interruption of descending supraspinal input on alpha motor neurons and interneurons, causing a transient dysfunction of the lower motor neuron (Smith and Jeffery 2005, de Lahunta and Glass 2009). This phenomenon has important implications because it may lead to erroneous neuroanatomic localisation, and therefore erroneous diagnostic investigations.

The most common site for cervical disc extrusion is the C2-C3 intervertebral disc in small dogs and C6-C7 in larger dogs (Cherrone and others 2004) but recent studies have reported C5-C6 and C6-C7 as the most commonly affected IVD spaces among all dogs with cervical disc herniation (Ryan and others 2008, Hillman and others 2009). In this study the most common site was the C4-C5 IVD and there was no distinction between small and large dogs. This may be due to the small population in this study.

Two dogs had a neurolocalisation at C6-T2 spinal cord segments and in both dogs the IVD affected was at C3-C4. A recent study (Forterre and others 2008) showed that withdrawal reflex in dogs with a cervical disc herniation is not reliable for determining the affected site and that a decreased withdrawal does not always indicate a lesion from C6 to T2 spinal cord segments. Dogs in which the lesion is clinically localised to the C6-T2 spinal cord segment should have imaging (radiographs, computed tomography or MRI) that includes the C1-T2 spinal cord segments in order to investigate the potential underlying cause of the clinical presentation.

Diagnosis of the HNPE was suspected at the time of surgery in seven dogs by the identification of the white, opaque and gelatinous disc material within the vertebral canal. Recent reports in dogs (Konar and others 2008, Kamishina and others 2010) described canine "discal cysts" affecting the cervical spine with similar clinical signs and MRI findings to the lesions in this study. All the cervical "discal cysts" cases reported in veterinary medicine were older than six years of age and had an acute onset of clinical signs (Konar and others 2008, Kamishina and others 2010) which is similar to our findings. None of the reported cervical "discal cysts" had a histopathological evaluation, a capsule could not be recognised in any of the cases that underwent cervical surgery, and the diagnosis was made based only on MRI features (Konar and others 2008). Discal cysts in humans have been described only affecting the lumbar spine and consist of a ventrolateral extradural cyst attached to the herniated lumbar disc with a thick fibrous capsule (Lee and others 2006). Magnetic resonance features in human beings with discal cyst are mainly characterised by a ventrolateral extradural cyst attached to a lumbar disc with a rim of low signal intensity on T2W that reflect fibrosis in the cystic capsules (Lee and others 2006). These MR imaging features differ from our findings. Moreover discal cysts in human beings do not cause spinal cord contusion or acute onset of clinical signs apart form radiculopathy (Dasenbrock and others 2010). Discal cyst is a progressive condition, needing time to develop and causing soft compression of the spinal cord (Chiba and others 2001). All the dogs presented in this study developed acute onset of clinical signs with often spinal cord contusion, which cannot be explained with a progressive cyst formation. Therefore based on the acute clinical presentation, imaging features, and intraoperative findings we believe that the dogs included in our study were not affected by "discal cysts" as reported in human medicine. None of the presented cases had histopathological evaluation of the extruded material. A potential source of weakness in this study is the lack

of histological confirmation but due the consistency between the imaging findings (location in relation to the disc space, reduced volume of hydrated nucleus pulposus, material intensity) and the gross surgical findings (location and physical nature of the material) histopathology was not considered necessary at the time of surgery. Further studies with histological evaluation may be necessary to confirm our findings.

Three dogs had mild contrast enhancement of the extradural compressive material but this is a common finding on MRI of dogs with IVD extrusion and should not be interpreted as a specific sign of a mass lesion (Suran and others 2011).

Both medical and surgical treatment can be used in the treatment of cervical disc herniation in dogs (Sharp and Wheeler 2005). In this study all dogs presented with severe neurological dysfunction and the therapy was selected based on the severity of the spinal cord compression on MRI. Dogs presenting with mild spinal cord compression underwent conservative management and regained ambulatory status in less than 48 hours, while the median time for dogs that underwent surgery to regain ambulatory status was five days. This discrepancy in recovery times is most likely due to less severe clinical signs and mildest degree of SCC in dogs selected for conservative management. In human literature, there are few reports with spontaneous regression of herniated cervical disc (Song and others 1999, Kobayashi and others 2002). The spontaneous regression has been related to the chemical inflammatory reaction on the extruded disc material and neovascularisation, which are thought to be required for phagocytosis (Haro and others 2002). In this study none of the dogs that underwent conservative management had a follow-up MRI in order to assess the affected area, but based on their clinical progress, the spontaneous regression of the hydrated nucleus pulposus extrusion is likely to have occurred.

The prognostic factors that predict recovery in surgically treated dogs with non-ambulatory tetraparesis secondary to cervical disc extrusion have been reported to be small body size and regaining ambulatory status within 96 hours postoperatively with full recovery rates of 58 to 62% (Waters 1989, Hillman and others 2009). In this study all surgically treated dogs but one had a successful outcome. The median bodyweight was 18 kg and the median time for dogs that underwent surgery to regain ambulatory status was five days, therefore these reported prognostic factors may not necessary apply to acute, compressive suspected HNPE extrusion. A study with a larger population and statistical analysis is required to confirm this finding.

In conclusion, compressive myelopathy caused by acute suspected HNPE has not been reported previously in dogs. Magnetic resonance imaging features are characteristic and show nuclear material of hydrated signal intensity immediately above the affected disc space, often with a "seagull" shape on transverse images. Even though neurological deficits can be severe on presentation, the outcome is favourable. The presence of compressive HNPE may indicate that another pathophysiological basis rather that the well-known Hansen type I degenerative changes causes the extrusion. More clinical studies, biomechanical analysis of the cervical spine and histological evaluation of the residual intervertebral disc and the extruded material may be necessary to better understand the pathophysiology behind different type of intervertebral disc extrusions.

Conflict of interest

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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