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Clinical Characteristics of Dogs with Progressive Myelomalacia Following Acute Intervertebral Disc Extrusion

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Background: Progressive myelomalacia (PMM) is a catastrophic disease associated with acute intervertebral disc extrusion (IVDE). Published data on the clinical characteristics of this disease are limited.

Objective: To describe the onset and progression of clinical signs of PMM in a large case cohort.

Animals: Fifty-one dogs, 18 with histopathologically confirmed PMM, 33 presumptively diagnosed based on clinical signs and diagnostic imaging.

Methods: Retrospective study. Dogs with confirmed IVDE and either a histopathologic diagnosis of PMM or a high clinical suspicion were identified by medical record search. Data on nature and progression of signs were extracted.

Results: Twenty-four of 51 dogs were Dachshunds. T12–T13 was the most common site of disc extrusion (12 of 56), and 18 of 55 of mid-to-caudal lumbar discs (between L3 and L6) were affected. Onset of PMM signs ranged from present at first evaluation (17/51) to 5 days after presentation, with 25 of 51 cases developing signs within 48 hours. Progression of signs from onset of PMM to euthanasia or death, excluding 7 cases euthanized at presentation, ranged from 1 to 13 days with 23 being euthanized within 3 days. Nonspecific systemic signs were documented in 30 of 51 dogs.

Conclusion and Clinical Importance: The majority of dogs developed PMM within 2 days of presentation and was euthanized within another 3 days. However, onset can be delayed up to 5 days after presentation with progression to euthanasia taking as long as 2 weeks. Mid-to-caudal lumbar discs might be associated with an increased risk of PMM.

Key words: Ascending-descending myelomalacia; Intervertebral disc disease; Spinal cord injury.

Progressive myelomalacia (PMM), also called ascending-descending myelomalacia, is a fatal disease characterized by progressive ascending and descending necrosis of the spinal cord after an acute intervertebral disc extrusion (IVDE).[1–3] The condition was first described histopathologically in 1972[1] and is characterized by severe thrombosis and hemorrhage within the spinal cord,[1,4] but its pathophysiology remains poorly understood.[3–5]

The clinical signs indicative of PMM reflect the involvement of extensive regions of the spinal cord gray matter, producing coincidental lower motor neuron signs in the pelvic limbs and cranial advancement of the cutaneous trunci muscle reflex (CTMR).[6] As the disease progresses, loss of anal and abdominal tone, paralysis of the thoracic limbs and of the respiratory muscles develops. Most dogs are humanely euthanized before respiratory failure results in their death.[3] The frequency of PMM ranges between 11 and 17.5% of dogs presenting with complete sensorimotor loss from spinal cord injuries (paraplegic with loss of pain perception in pelvic limbs and tail) following IVDE.[6–8] However, it may differ between breeds as the rate of PMM was reported to be as high as 33% in French Bulldogs with paraplegia and loss of pain perception.[9]

While there are several published studies on the histopathologic lesions, risk factors for its development, diagnostic, and imaging characteristics of PMM, to date, there is no large-scale retrospective study describing its clinical characteristics.[4,10–15] The goal of this study was to describe the clinical signs associated with PMM, including their time of onset and their rate of progression in a large case cohort.

Abbreviations:

CSF	cerebrospinal fluid
CTMR	cutaneous trunci muscle reflex
IVDE	intervertebral disc extrusion
MRI	magnetic resonance imaging
PMM	progressive myelomalacia
SCI	spinal cord injury

Material and Methods

Case Selection

The medical records of dogs diagnosed with IVDE at North Carolina State University Veterinary Hospital from 1998 to 2016 were searched to identify cases with either a definitive diagnosis of PMM obtained by histopathology or a presumptive diagnosis of PMM. A presumptive diagnosis of PMM was made based on a combination of specific clinical signs that demonstrated a mismatch between neuroanatomic diagnosis and site of IVDE and clinical progression. All dogs were paraplegic at admission or became paraplegic during hospitalization with no pain perception

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in both pelvic limbs and the tail. Additional clinical signs considered consistent with PMM included complete loss of pelvic limb reflexes (patellar, withdrawal) and perineal reflex when inconsistent with the site of disc extrusion, loss of abdominal tone, cranial advancement of the CTMR during hospitalization or a CTMR caudal border more than two vertebral levels cranial to the site of disc extrusion, and potentially progression to tetraparesis. Loss of pelvic limb spinal reflexes was suspected to be the result of PMM and was differentiated from spinal shock when there was loss of both the patellar and withdrawal reflex at presentation that persisted during hospitalization, in cases with disc extrusions cranial to the lumbosacral intumescence (cranial to the L5 spinal cord segment or L4 vertebra[16]), or when they were lost during hospitalization. The age of the dog was taken into consideration to avoid including dogs with potential age-related patellar reflex loss.[17] Reduced or absent pelvic withdrawal reflexes alone, with a normal patellar reflex, were not considered as a sign of PMM as this finding could occur secondary to spinal shock.[18] Dogs that were euthanized the day of admission before clinical progression could be established either had a histopathologic diagnosis of PMM or clearly demonstrated a combination of neurologic findings that established a wide lesion extent along with MRI evidence of PMM.

Imaging findings consistent with PMM were also taken into account. Magnetic resonance imaging (MRI) features considered suggestive of PMM included parenchymal hyperintensity extending more than six times the length of the vertebral body of L2 on sagittal T2-weighted images associated with an IVDE[11] as well as complete loss of the cerebrospinal fluid (CSF) signal over the entire thoracolumbar spinal column on the half-Fourier acquisition single-shot turbo spin-echo (HASTE) sequence when performed. A ratio of the length of CSF attenuation to the length of the vertebral body of L2 ($CSF:L2_{HASTE}$) $\geq$7.4 was considered highly suspicious for PMM.[16]

Dogs were excluded as presumptive cases if there was no imaging performed to confirm an acute IVDE. Dogs were also excluded if the progression of the signs stopped and they recovered or if they were discharged and the follow-up was insufficient to determine whether they had recovered.

Clinical Information

The following information was extracted from the medical record: age, breed, sex, weight, time from onset of clinical signs of IVDE (including pelvic limb weakness, gait deficits, reluctance to jump, kyphosis, or pain) to presentation to NCSU-VTH, time from loss of ambulation to presentation at NCSU-VTH (in hours), time from loss of motor function to development of signs suggestive of PMM, time from presumptive onset of PMM to death or euthanasia, systemic signs possibly associated with PMM, cerebrospinal fluid analysis, imaging findings including site of extruded intervertebral disc, surgical findings, and histopathologic findings. Time periods were expressed in hours when possible and categorized into $\leq$12 hours, 12 to 24 hours, $\leq$24 hours, 24 to 48 hours, $\leq$48 hours, 48 to 72 hours, and >72 hours. Summary data were prepared for these parameters.

Results

Fifty-one dogs met the inclusion criteria. Five additional dogs had consistent clinical signs such as cranial advancement of the CTMR or loss of their spinal reflexes during hospitalization, but were excluded due to halting of progression of the disease. Two of these dogs had repeat MRI showing a focal intraparenchymal lesion cranial to the site of surgery suggestive of a vascular event. Both dogs recovered. A third one recovered but did not have repeat imaging. The 2 last dogs did not have repeat imaging and were lost to follow-up so it was not possible to determine whether they had recovered. Of the included cases, 18 of 51 had a histopathologic diagnosis of PMM, 17 of 51 dogs had clinical signs and MRI findings consistent with PMM, and 16 of 51 dogs just had consistent clinical signs.

Signalment

The majority of the dogs was Dachshunds (24 of 51). Other breeds included Cocker Spaniel (3 of 51), Pekingese (3 of 51), Corgi (2 of 51), and one of each of the following: Beagle, French Bulldog, Basset-Hound, Papillon, Golden Retriever, Yorkshire Terrier, Affenpinscher, Pomeranian, and Coton de Tulear. The remaining 10 dogs were mixed-breed. Breed representation in the histopathologically confirmed subset was comparable, with Dachshunds representing half the cases (9 of 18). Other breeds included Cocker Spaniels (3 of 18) and one each of the following: Beagle, Corgi, French Bulldog, Pekingese, Yorkshire Terrier, and Mixed-breed.

The median age in the whole group and the histopathologically confirmed subset was 5 years (range 2–14). The median weight was 7.5 kg (range 3.4 kg-30.8 kg) for the whole group and 7.9 kg (range 3.4 kg-18.4 kg) for the histopathologically confirmed subset. Males and females were equally represented with 26 females (22 spayed, 4 intact) and 25 males (18 neutered, 7 intact) in the whole group. Similar findings were noted in the histopathologically confirmed subset with 9 females (8 spayed, 1 intact) and 9 males (7 neutered, 2 intact).

Imaging Findings

Computed tomography (CT) scans were obtained alone in 23 of 51 cases and combined with a myelogram in 5 of 51 cases. One case had a myelogram only. Although the CT, CT/myelogram, and myelogram alone allowed visualization of the site of disc herniation in all dogs, none of these imaging modalities revealed findings suggestive of myelomalacia as previously reported.[19]

Magnetic resonance imaging was performed in 20 of 51 dogs, 17 of which (85%) had features suggestive of PMM. Seventeen dogs (85%) had a $CSF:L2_{HASTE}$ $\geq$7.4. Of these, 9 of 20 dogs (45%) showed T2 hyperintensities $\geq$6 times the length of the vertebral body of L2.

Two dogs (2 of 20) had both a MRI and a CT scan performed. One of them, a French Bulldog, had an MRI to assess the integrity of the spinal cord parenchyma and the presence of a compressive lesion combined with a CT scan to better characterize congenital vertebral anomalies. The other dog had an MRI on initial presentation. Following worsening neurologic status and loss of pain perception 4 days after presentation, a

CT scan was performed and revealed a second disc herniation requiring a second surgical intervention.

One dog had two MRIs. This dog lost pain perception in the pelvic limbs and tail 48 hours after imaging and surgery. A second MRI was performed, and this time revealed features consistent with myelomalacia. Four cases did not undergo any imaging, but a disc extrusion was diagnosed at necropsy.

Sites of Disc Extrusion

A total of 56 disc extrusions were reported as 5 dogs had 2 sites involved. The most common sites of disc extrusion were T12–T13 (12 of 56), L4–L5 (10 of 56), T13–L1 (9 of 56), and L2–L3 (9 of 56). Other sites were T11–T12 (4 of 56), L3–L4 (4 of 56), L5–L6 (4 of 56), L1–L2 (3 of 56), and T10–T11 (1 of 56). In the histopathologically confirmed subset, 18 disc locations were reported with the most common location being T12–T13 (6 of 18). Other disc locations included L4–L5 (3), T13–L1 (2), L2–L3 (2), L5–L6 (2), T11–T12 (1), L1–L2 (1), and L3–L4 (1).

The disc extrusion was reported to be focal in the majority of the dogs (41 of 56 for all dogs and 13 of 18 for histopathologically confirmed subset). It was considered extensive, that is, with disc material causing spinal cord compression over 2 or more sites based on imaging for the other dogs (15 of 56 for all dogs and 5 of 18 for the histopathologically confirmed subset).

Cerebrospinal Fluid Analysis

A lumbar CSF tap was performed in 12 of 51 dogs. The median white blood cell count was 50/μL (range 2–650; reference interval 0–5/μL), the median red blood cell count was 5,757/μL (range: 127–75,000; reference 0/μL), and the median protein concentration was 36.6 mg/dL (range: 17.8 – 94.8; reference: less than 40 mg/dL). A neutrophilic pleocytosis was present in 5/12 cases, mixed pleocytosis in 3 of 12, and mononuclear pleocytosis in 1 of 12. Macrophages with intracellular myelin debris were seen in one case. Albuminocytological dissociation was present in 1 of 12. The specimen was too small to be analyzed in one case, and the cytology findings were not reported in one case.

Surgical Treatment

Surgery was performed in 40 of 51 cases. The appearance of the spinal cord during surgery was reported in 29 cases and was described as discolored/bruised in 20 cases, swollen only in 7 cases, and normal in 2 cases.

Histopathologic Findings

The necropsy reports were reviewed in 18 of 51 dogs, and the findings were all consistent with PMM. Acute disc extrusion was reported in 9 dogs. The 9 other dogs for which there was no report of acutely extruded disc material had all previously undergone surgery.

Onset and Progression of Neurologic Signs

The time from onset of signs of disc extrusion to presentation ranged from 2 hours to 30 days (Table 1). Most dogs showed clinical signs for ≤48 hours before presentation (34 of 51). The time from onset of clinical signs to loss of ambulation ranged from 0 to 29 days. This time could not be established for 4 cases. More than half of the dogs (31 of 47) lost the ability to walk within 12 hours following the onset of clinical signs. For the remaining 16 dogs, 8 lost ambulation within 24 hours, 5 within 48 hours, and 3 >72 hours after onset of signs. The time from loss of ambulation to presentation ranged from 2 hours to 5 days. Three dogs were still ambulatory at presentation. Most dogs presented within 24 hours following loss of ambulation (34 of 48). The 3 dogs that were ambulatory at presentation started to show clinical signs <24 hours before presentation (1), <48 hours (1), and 7 days before presentation (1). These 3 dogs all lost the ability to walk within 24 hours after presentation. The time from presentation to onset of signs of PMM ranged from 0 hours (ie, the signs were present at presentation) to 5 days. One third of the dogs had signs suggestive of PMM at presentation (17 of 51). The time of onset of signs for the other dogs is reported in more detail in Table 2.

Fifty dogs were euthanized, and the remaining one died at home. The time from presentation to death or euthanasia ranged from 0 (ie, the dog was euthanized on presentation) to 17 days. Seven dogs were euthanized at presentation. Of the remaining 44 dogs, a similar number was euthanized ≤72 hours (23) and >72 hours (21) following presentation. The time from onset of signs of PMM to death or euthanasia was determined for the 31 dogs that developed signs of PMM while hospitalized (see Table 2). This ranged

Table 1. Timeline of progression of clinical signs of IVDE.

	Number of Dogs
Time from onset of signs of IVDE to presentation	
≤12 hours	5
12–24 hours	19
24–48 hours	10
48–72 hours	5
>72 hours	12
Time from onset of signs to loss of ambulation	
Peracute	11
≤12 hours	20
≤24 hours	8
≤48 hours	5
>48 hours	3
Not reported	4
Time from loss of ambulation to presentation	
≤12 hours	17
12–24 hours	17
24–48 hours	5
48–72 hours	4
>72 hours	5
Ambulatory on presentation	3

Table 2. Timeline of progression of clinical signs of PMM and time to death or euthanasia.

	Number of Dogs
Time from presentation to onset of signs of PMM	
Signs present at presentation	17
≤12 hours	3
12–24 hours	7
24–48 hours	15
48–72 hours	2
>72 hours	7
Time from presentation to death or euthanasia	
At presentation[a]	7
≤24 hours	7
≤48 hours	5
≤72 hours	11
>72 hours	21
Time from onset of PMM to euthanasia[a]	
12–24 hours	10
24–48 hours	9
48–72 hours	5
>72 hours	7

[a]Determined in only 31 dogs.

from less than 12 hours (the dog was euthanized the same day as the onset of signs) to 13 days. The dog that survived for 17 days following presentation (ie, 13 days following onset of signs of PMM) had sudden loss of pelvic limb reflexes 5 days after surgery, and then signs remained static until sudden deterioration 12 days later.

Onset and Progression of Signs of PMM

Seventeen dogs (17 of 51) had neurologic findings suggestive of PMM at presentation (9 of these dogs were histopathologically confirmed). All these dogs were paraplegic with no pain perception in their pelvic limbs or tail. The signs of PMM were weak to absent patellar reflexes and weak to absent anal tone and perineal reflex in cases with a disc extrusion located cranial to level of the lumbosacral intumescence (8), CTMR off at least two disc spaces cranial to the site of disc extrusion (11), loss of abdominal tone (3), difficulty maintaining sternal recumbency (2), and thoracic limb involvement (2). All these dogs were showing a combination of at least 2 of these signs except for 2 with only an abnormal CTMR and 1 with loss of pelvic limb and perineal reflexes.

Forty-two dogs (42/51) were paraplegic at presentation. The remaining 9 dogs became paraplegic 2 hours to 4 days after presentation with the majority (5 of 9) within the first 24 hours after presentation. Thirty-nine (39 of 51) cases had lost pain perception on presentation. The 12 dogs that presented with pain perception lost it between 2 hours and 4 days after presentation, half of them (6 of 12) within the first 24 hours after presentation.

The CTMR was noted to be intact on presentation in 19 of 51 dogs and at the level of the lesion in 18 of 51 dogs. It was reported to have a caudal border at least two disc spaces cranial to the site of disc extrusion in 11 of 51. For the 3 remaining dogs, it was not reported or unclear. Two dogs with a normal CTMR and a CTMR caudal border at the lesion were euthanized on presentation, both had a histopathologic diagnosis of PMM. In the remaining 35 dogs that had either a normal CTMR or a CTMR at the level of the lesion, the timeline for cranial advancement ranged from 24 hours to 16 days with advancement occurring in 8 of 35 within 24 hours, 12 of 35 within 48 hours, 6 of 35 within 72 hours, 9 of 35 >72 hours following presentation.

Decreased to absent patellar reflexes not consistent with the site of disc extrusion was noted at presentation in 8 dogs. These dogs were all younger than 10 years (the oldest was 6 years old) making age-related loss of patellar reflexes less likely.[17] Half of them (4 of 8) had histopathologic confirmation of PMM and had an onset of signs more than 48 hours before presentation thus making spinal shock less likely. The other 4 dogs had persistent loss of their spinal reflexes and showed progression of signs consistent with PMM throughout their hospitalization. Fifteen additional dogs lost their pelvic limb reflexes within 5 days after presentation, with 3 within 24 hours, 7 within 48 hours, 1 within 72 hours, and 4 >72 hours.

The perineal reflex and anal tone were abnormal on presentation in 9 dogs (4 histopathologically confirmed) and were lost during the hospitalization time in 12 others, 2 within 24 hours, 6 within 48 hours, 2 within 72 hours, and the last 2 >72 hours following presentation. There was not enough information related to progression of pelvic limb reflexes in the medical record for the other dogs.

Four cases had decreased to absent abdominal tone on presentation. Sixteen other cases lost their abdominal tone between 24 hours and 16 days, with 3 within 24 hours, 3 within 48 hours, 2 within 72 hours, and 8 >72 hours. In the remaining cases, loss of abdominal tone was not noted in the medical record.

The respiratory pattern was considered abnormal (ie, decreased chest wall excursion, tachypnea, or dyspnea) in 18 of 51 cases. One of these dogs was ultimately diagnosed with aspiration pneumonia. For the other dogs, the time for onset of respiratory signs ranged from 0 hours (at presentation) to 13 days with 2 dogs showing signs on presentation, 1 within 24 hours, 2 within 48 hours, 6 within 72 hours, and 6 >72 hours.

Two dogs presented with difficulty maintaining sternal recumbency, 1 had a histopathologic diagnosis while the other one had a cervical and lumbar MRI, revealing an acute disc herniation at T12–T13 and significant spinal cord changes consistent with PMM. Nine additional dogs developed difficulty maintaining sternal recumbency within 8 days of hospitalization (most dogs, 5 of 9, within 72 hours).

Twenty-three dogs (23 of 51) presented with or developed thoracic limbs paresis or proprioceptive deficits. The time for development of thoracic limb deficits ranged from 0 hours (deficits present at presentation) to 10 days with 2 with signs of thoracic limb involvement

at presentation, 2 within 24 hours, 3 within 48 hours, 5 within 72 hours, and 11 >72 hours.

Altered mentation was reported in 6 of 51 dogs. Three dogs became stuporous, 1 showed episodes of stargazing, one became comatose with seizure-like episodes, and one was obtunded. All the dogs showed these signs within 7 days after onset of signs of PMM. All 6 dogs with alteration of their mentation were also tetraparetic.

Systemic Signs

More than half of the dogs (30 of 51) had systemic abnormalities documented. These included hypothermia (ie, rectal temperature lower than 99°F or 37.2°C) in 15 of 51 dogs, fever (ie, rectal temperature higher than 102.5°F or 39.1°C) in 7 of 51 dogs, and diffuse hyperesthesia (ie, not localized to the spine) in 9 of 51 dogs.

Discussion

This retrospective study describes the clinical characteristics and timeline of progression of PMM in 51 dogs after IVDE. The majority of dogs was Dachshunds. The most common site of disc herniation was T12–T13, but a relatively high number of mid-to-caudal lumbar discs (L3–L6) were also noted. Most dogs developed signs of PMM within 2 days of presentation and progressed to euthanasia within 4 days of onset of signs. However, onset was delayed up to 5 days after presentation with progression to death taking almost 2 weeks. Nonspecific systemic signs such as abnormal rectal temperature or mentation change that could be attributed to PMM were reported in more than half of the dogs.

The gold standard for diagnosis of PMM is necropsy confirmation, which was achieved in about a third of our cases. Unfortunately, many owners decline necropsy so we included dogs with clinical signs that reflected the progressive expansion of myelopathy, due to progressive involvement of spinal cord gray matter specifically. The similarity of results in the whole case cohort and the subset with necropsy confirmation suggests our diagnostic criteria were adequate. An antemortem bedside diagnostic test for PMM has not yet been developed, although measurement of serum levels of glial fibrillary acid protein (GFAP), a major constituent protein of mature astrocytes, using an ELISA test is highly specific (97.7%), with lower sensitivity (75%).[13] This test is unfortunately not available as a rapid turnaround test.

The dog breeds represented reflect the breeds reported in large studies of acute IVDE.[7,20–22] Similarly, the most common sites of disc extrusion are comparable to these studies.[7,20,23,24] However, a high proportion of mid-to-caudal lumbar and more specifically L4–L5 disc extrusions was noted with 35% (18 of 51) of dogs having an extrusion between L3 and L6 compared with 16% (11 of 70) reported in a similar population of dogs with negative pain perception.[6] While one of our criteria for a presumptive diagnosis of PMM was the absence of patellar reflexes, this sign was only considered a potential sign of PMM if the disc location did not correlate with the neuroanatomic diagnosis, and we relied on a combination of signs such as a CTMR cutoff at least two disc spaces cranial to the site of extrusion and progressive deterioration of the neurologic status, making misdiagnosis unlikely. In addition, a relatively high number of mid-to-caudal lumbar discs were also noted in the histopathologically confirmed group.

Our findings of a high proportion of mid-to-caudal lumbar disc herniations corroborate the findings of a recent study of 13 cases of PMM that evaluated risk factors associated with this condition where L5–L6 site of IVDE was associated with an increased risk of developing PMM.[14] It has been hypothesized that IVDE in the caudal lumbar spine could damage a large spinal artery located there, causing a larger ischemic injury and triggering the development of PMM.[3,14] It is also possible that IVDE in the mid-lumbar area affects the lumbosacral intumescence disrupting a larger area of gray matter, causing more severe secondary injury,[25] again increasing the chance of triggering PMM. Neither of these theories truly accounts for the stepwise progression of necrosis along the spinal cord. Another group postulated that cranial and caudal propagation of necrotic debris along the central canal could cause further hemorrhagic lysis of the spinal cord tissue in segments remote from the initial injury, resulting in PMM.[4,26] Endothelin overexpression has also been implicated.[5] Finally, Griffiths speculated that PMM results from multilevel injury to the vasculature by extensive disc extrusions.[1] However, our data demonstrate that PMM is associated with both focal and extensive disc extrusions.

One study suggested that an area of hyperintense signal within the spinal cord longer than 6 times the length of the vertebral body of L2 on sagittal T2-weighed magnetic resonance images was suggestive of myelomalacia.[11] This finding was only observed in 45% of the dogs that underwent an MRI. Therefore, the absence of extensive hyperintense signal in the cord on sagittal T2W images does not preclude the development of PMM. The presence of a CSF:$L2_{HASTE}$ $\geq$7.4 times the length of the vertebral body of L2 was reported to have a sensitivity of 100% and a specificity of 75% to diagnose myelomalacia.[16] It was noted in 85% of our cases and is a more reliable MRI criterion for the suspicion of PMM than T2 hyperintensity, but, again, the absence of this finding does preclude the development of PMM. The 3 dogs with a ratio CSF:$L2_{HASTE}$ <7.4 were all imaged within 12–24 hours following loss of ambulation and developed signs suggestive of PMM 2–5 days after presentation. The timing between imaging and onset of signs of PMM is likely to be important to detect these changes as previously reported.[14] One dog in our group had a first MRI performed within 24 hours following presentation and had a second MRI performed 48 hours later due to acute worsening of its neurologic status with loss of pain perception, decrease in pelvic limb reflexes, abdominal, and anal tone. The MRI revealed patchy intraparenchymal T2-hyperintensity extending from T8 to the conus medullaris. These changes were considered consistent with myelomalacia.

The dog was euthanized the next day due to progression to tetraplegia and obtundation. Therefore, repeating the MRI in dogs who developed signs of PMM after the first imaging modality might increase the likelihood of finding changes suggestive of the disease.

Cerebrospinal fluid analysis was performed in a limited number of cases. The results were quite variable, could be attributed to the inflammatory changes associated with IVDE as previously reported, and were not necessarily characteristic of PMM.[27] None of the markers previously reported to be indicative of the severity of the injury were analyzed.[28–31] The majority of dogs underwent surgery. When the appearance of the spinal cord during surgery was reported, it was abnormal in 93% of dogs. While this is a subjective assessment, comparison of reported spinal cord appearance in an appropriate control population of dogs undergoing hemilaminectomy for IVDE will be of interest.

In our study, the majority of dogs presented paraplegic with absent pain perception. However, 12 of 51 dogs (25%) still had pain perception at the time of presentation, and 9 had motor function with 3 being ambulatory. Most prior publications reporting PMM have focused on dogs that presented with no pain perception.[6,32] A more recent study, however, reported an incidence of PMM in dogs presented with nonambulatory paraparesis (grade 3) or paraplegia with intact pain perception (grade 4) of 0.6% and 2.7%, respectively.[14] This study and our data suggest that it is important to discuss this complication with owners at the time of presentation of dogs with suspected thoracolumbar IVDE regardless of the initial neurologic status of the dog.

A majority of dogs (39 of 47) lost the ability to walk within 24 hours of onset of signs of IVDE. Among these dogs, 28% (11 of 39) had a peracute onset of loss of ambulation, as they were found acutely unable to walk by the owner with no preceding signs. These data corroborate the findings of Balducci et al.[14] that dogs with a duration of clinical signs of less than 24 hours were 3.54 times more likely to develop PMM than dogs with signs for more than 24 hours. This suggests that PMM is commonly associated with a rapid deterioration in the neurologic status following IVDE, which could be related to the severity of the initial contusive spinal cord injury. However, this is not always the case, as 16 dogs had more gradual onset with a small number developing signs over more than 3 days. As such, PMM should be considered even in dogs with gradual onset of paralysis. Because of the variability in onset of signs of PMM, it is difficult to provide any recommendation for surgeons on whether or not waiting to perform surgery would help to screen out PMM cases better and avoid an invasive and costly procedure on dogs with a grim prognosis. However, the presence and evolution of clinical signs of PMM as described here should help to identify these cases at the first opportunity.

Most of the dogs developed signs suggestive of myelomalacia within 48 hours of presentation, and it is important to note that a third of them had signs at the time of presentation. The more subtle early signs of PMM can be easy to overlook in the rush to complete imaging and surgery in these emergent cases. By contrast, a small number of dogs had a later onset of signs with some dogs developing signs of PMM up to 5 days after presentation. The average duration from onset of signs to death or euthanasia was 4 days but some dogs had delayed progression. Indeed, 1 dog showed onset of signs 5 days after presentation, then remained static for 7 days. The reason for the variability in progression of signs is unknown but may reflect the vascular nature of this disease.[12] A recent study described intramedullary as well as subdural hemorrhages and speculated that spinal cord debris admixed with CSF accumulating in the central canal were propelled cranially and caudally due to the increased intramedullary pressure causing longitudinal spreading of the necrotic tissue and subsequent hemorrhagic lysis of adjacent spinal cord segments.[4] Sudden spread of debris might explain the stepwise and intermittent progression of signs.

We excluded 5 dogs that initially showed signs suggestive of PMM in which the signs failed to progress and 3 of these dogs recovered. We chose not to include the other 2 cases because the lack of long-term follow-up meant it was impossible to tell whether the signs observed in these dogs were due to lack of further progression of PMM or whether these dogs had suffered a separate event causing acute change in their neurologic status as confirmed in 2 others and previously reported.[33]

Systemic signs not usually appreciated in dogs after spinal surgery were reported in a majority of cases. These signs such as altered mentation and temperature could also be attributed to brain involvement. This could be explained by PMM being triggered by systemic disease, or could be a consequence of the massive changes occurring within the spinal cord including free radical and vasoactive substance release diffusing toward the brainstem and the cerebrum.[9] The profound vascular thromboses within the spinal cord characteristic of this disease[1,5,12] could result from or be exacerbated by a pro-inflammatory or procoagulable state set up by the initial SCI that would secondarily spread to the rest of the central nervous system but also systemically, potentially affecting other organs.

Our study had limitations primarily because of its retrospective nature. Only 18 of 51 dogs had a definitive histopathologic diagnosis of PMM. However, similar findings were noted when looking at the confirmed and presumptive subsets suggesting that results for all the dogs included are likely reflective of the clinical characteristics of the disease. The data collected for all dogs were extracted from their medical record and were therefore reliant on details recorded by the clinician at the time. As such, specific details on the presence of signs such as loss of abdominal tone might not have been entered, and the precise chronology of progression may be inaccurate. Some dogs were euthanized on presentation or early in the course of the disease without repeating the MRI. It is possible that for some of them, the progression of the disease would have eventually stopped and the animal could have survived.

Conclusion

In this study, the majority of dogs that developed PMM was Dachshunds, and they had signs suggestive of PMM at presentation or developed them within 2 days of presentation. While the majority deteriorated and were euthanized within 4 days following onset of PMM, onset can be delayed as long as 5 days after presentation with protracted progression taking up to 2 weeks. The risk period for development of PMM is therefore longer than typically understood. Dogs with mid-lower lumbar disc extrusions seem to be at increased risk of developing the disease.

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