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Signalment, Clinical Presentation, and Diagnostic Findings in 122 Dogs with Spinal Arachnoid Diverticula

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Background: Most information about spinal arachnoid diverticula (SADs) in dogs has been retrieved from relatively small case series. The aim of this study was to describe this disease in a larger number of dogs.

Objectives: Description of the signalment, clinical presentation, and imaging findings of a large number of dogs with SADs.

Animals: One hundred and twenty-two dogs with SADs.

Methods: Retrospective case series study. All medical records were searched for a diagnosis of SAD. The diagnosis was made based on myelography, computed tomography myelography (CT-m), or magnetic resonance imaging (MRI).

Results: In the 122 dogs, 125 SADs were identified. Sixty-five were located in the cervical region and 60 in the thoracolumbar region. A higher body weight was significantly associated with a cervical localization of the SAD ($P < .001$). Ninety-five dogs were male and 27 dogs were female. Male dogs were significantly overrepresented ($P < .0001$). The most commonly affected breed was the Pug dog. Previous or concurrent spinal disorders, in the near proximity of the diagnosed SAD, were seen in 26 dogs. Eight of 13 French Bulldogs and 7 of 21 Pug dogs with SADs had a previous or concurrent spinal disease, whereas other spinal disorders occurred in only 1 of 17 Rottweilers with SADs.

Conclusions and Clinical Importance: Pug dogs and French Bulldogs might have a predisposition for SAD development. In a large percentage of these dogs, a concurrent spinal disorder, which might predispose to SAD formation, was diagnosed. The high prevalence in male dogs warrants further investigation.

Key words: Dog; Spinal arachnoid cyst; Spinal arachnoid diverticulum; Spinal cord.

Spinal arachnoid diverticula (SADs) are focal dilations of the subarachnoid space, which can lead to a progressive, compressive myelopathy. Formerly, the term spinal arachnoid cyst was commonly used, but this is currently considered a misnomer as these lesions lack an epithelial lining.[1]

SADs have been described in people, 1 horse, and occasionally in cats and dogs.[1–6] Clinical signs can vary from a mild-to-severe generalized proprioceptive ataxia with fecal, urinary incontinence, or both. The most characteristic clinical presentation is a moderate generalized proprioceptive ataxia without any signs of discomfort. Although SADs can occur over the entire length of the vertebral column, cervical lesions are seen most commonly in large breed dogs while thoracolumbar lesions are more common in small breed dogs.[5] In agreement with studies in people, different etiologies have been suggested in dogs, including hereditary and congenital causes, biomechanical factors, and concurrent or previous spinal disorders, for example, intervertebral disk extrusion and protrusion or inflammatory disorders, such as meningo-myelitis.[1,5–8]

Although several studies have suggested an overrepresentation of Rottweiler dogs, little is known about the predisposition of other dog breeds.[1,6] Furthermore, most information about SADs originates from small case series or case reports. This currently hampers our further understanding of breed and sex predispositions, age distribution, variation in presenting clinical signs, and the prevalence of concurrent and possibly associated spinal abnormalities. This study describes signalment, clinical signs, and imaging findings in a larger group of dogs with SADs.

Abbreviations:

SAD	spinal arachnoid diverticulum
CT	computed tomography
CT-m	computed tomography myelography
MRI	magnetic resonance imaging
CSF	cerebrospinal fluid
TNCC	total nucleated cell count
IQR	interquartile range
PCR	polymerase chain reaction
FLAIR	fluid attenuated inversion recovery

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Materials and Methods

Dogs

Medical records and imaging findings of dogs with a diagnosis of SAD between January 2000 and March 2012 were reviewed retrospectively. The dogs were examined at the Centre for Small Animal Studies, Animal Health Trust, England (n = 60 dogs); Faculty of Veterinary Medicine, Ghent University, Belgium (n = 46); and the Royal Veterinary College, University of London, England (n = 16). Only dogs with complete medical records and imaging studies available were included. Information concerning age at onset and diagnosis, sex, body weight, duration of clinical signs, presenting clinical signs, and previous or concurrent neurologic diseases was collected from the medical records. For this study, only compressive spinal cord disorders near the location of the SAD, such as intervertebral disk extrusion or protrusion and vertebral malformations, inflammatory meningeal and spinal cord diseases, other cystic disorders, such as intracranial arachnoid diverticula and synovial cysts, and vascular disorders, such as fibrocartilaginous embolism nearby the localization of the SAD, were taken into account. To compare the occurrence of concurrent diseases between juvenile and adult dogs, adult dogs were defined as being 12 months or older. To take the different frequency of dog breeds in the hospital population into account, we calculated the ratio of SAD patients of a certain breed at each institution compared with the total number of dogs of this breed presented to that institution over the same time period. Breeds were excluded from this calculation, when only 1 or 2 dogs had been diagnosed with a SAD, as this can be incidental and does not necessarily reflect an overrepresentation especially in very unusual breeds. For 65 dogs, the results of cerebrospinal fluid (CSF) examination were available. In 19 dogs, CSF was obtained by cisternal puncture and in 22 dogs by lumbar puncture. In 2 dogs, both cisternal and lumbar punctures were performed. The site of CSF acquisition was not specified in the medical records of 22 other dogs. A total nucleated cell count (TNCC) below 5 cells/μL was considered normal for both cisternal and lumbar CSF samples. A total protein concentration below 30 mg/dL was considered normal for cisterna magna CSF samples, whereas a total protein concentration below 45 mg/dl was normal for lumbar CSF samples.[9]

Imaging Studies

All imaging studies were performed under general anesthesia. Anesthesia protocols varied among the dogs and among institutions. Diagnosis was based on myelography (n = 25), myelography in combination with computed tomography myelography (CT-m) (n = 13), magnetic resonance imaging (MRI) (n = 73), or a combination of myelography and MRI (n = 11). Myelography and CT-m were performed by cisternal or lumbar intrathecal injection of iohexol[a] contrast medium (0.2 mL/kg, maximal dose 10 mL). CT-m was performed with a 4-slice CT scanner.[b] MRI studies were performed by either a 0.2T[c] (n = 7) or 1.5T,[d,e] (n = 66) system. Eleven dogs underwent myelography first followed by MRI using a 0.2T (n = 1) or 1.5T (n = 10) imaging device. MRI protocols varied among patients, but all included T1- and T2- weighted sagittal and axial sequences of the region of interest.

Statistical Analysis

The relationship between localization of the SAD and the variables such as sex, age, and body weight was assessed using a logistic regression model with the different clinic populations as random effect and sex, age, and bodyweight as fixed effects. The response variable was probability of a cervical or thoracolumbar localization.

A binomial test was used to make comparisons between male and female dogs and between neutered and intact dogs. A Fisher exact test was used to compare the likeliness of a concurrent disease between adult and juvenile dogs and between different breeds and to compare the occurrence of clinical signs between the cervical or thoracolumbar localization. A *t*-test was performed to compare the age at onset between the different affected breeds and between male and female dogs.

Commercially available software[f] was used for all analyses and significance was defined as $P < .05$.

Results

Localization of SAD

A total of 122 dogs were identified. Because 3 dogs were diagnosed with multiple SADs, 125 SADs were included in this study. Sixty-five SADs (52%) involved the cervical region, whereas 60 SADs (48%) were found in the thoracolumbar region. Of the 3 dogs with multiple SADs, 1 dog had 2 isolated SADs involving thoracic spinal cord segments, and of the other 2 dogs, each had 1 cervical and 1 thoracic SAD. In dogs with a cervical SAD, the most common localization was C2-3 (n = 34), followed by C5-6 (n = 8). Other cervical localizations were less frequent. In dogs with thoracolumbar lesions, 8 dogs had a T13-L1, 7 dogs a T11-12, and 5 dogs a T9-10 lesion. Only 3 dogs had lumbar SADs (L2, L4-5, lumbosacral) (Fig 1). Other thoracolumbar localizations were less frequent. SADs extending over more than 2 vertebrae only occurred in 3 dogs. Two dogs had SADs from T1-L3 (Fig 2) and another dog had a SAD from T9-L5.

Multilobulated SADs occurred in 4 dogs, all located in the cervical region. One of these SADs was diagnosed at the C1-2 level in a Miniature Schnauzer with concurrent atlantoaxial instability, one at C3-4 in a Labrador Retriever and two at the C5-6 intervertebral disk space in a Rottweiler and a Dogue de Bordeaux.

Of all 125 SADs, 104 were located dorsally (83.2%), 8 ventrally (6.4%), and 5 on the left or right side lateral to the spinal cord (4.0%). In the dog with the lumbosacral SAD, the dural sac was dilated. 5.6% of SADs surrounded the spinal cord as an encircling structure (n = 7). This type of SAD was seen in 7 Rottweilers, 1 German Shepherd Dog, and 1 Bullmastiff. In 4 Rottweilers and in the German Shepherd Dog, this type of SAD occurred in the caudal cervical region, being located at C5-6 or C6-7 (Table 1). In 1 Rottweiler, this form of SAD was seen at the C2-3 space and in the Bullmastiff, at the level of the 5th thoracic vertebra.

Signalment

Of the 122 dogs, 95 dogs were male (78%) and 27 dogs were female. Sixty-eight dogs were male intact, 27 male neutered, 14 female intact, and 13 female

Table 1. Affected breeds with localization of the SAD.

	Total	Cervical	Thoracolumbar	Multiple
Pug dog	21	7	13	1
Rottweiler	17	15	2	0
French Bulldog	13	0	13	0
Bernese Mountain Dog	5	4	1	0
Dogue de Bordeaux	5	4	1	0
Labrador Retriever	5	5	0	0
West Highland White Terrier	5	0	5	0
Dachshund	4	2	2	0
Staffordshire Bullterrier	4	2	2	0
Boxer	3	2	1	0
German Shepherd Dog	3	2	1	0
Jack Russell Terrier	3	2	1	0
Mixed breed dog	3	1	2	0
Rhodesian Ridgeback	3	2	1	0
American Bulldog	2	2	0	0
Basset Hound	2	2	0	0
Chihuahua	2	0	1	1
Miniature Schnauzer	2	2	0	0
Shi Tzu	2	1	1	0
Alaskan Malamute	1	1	0	0
Bichon Frisé	1	1	0	0
Border Terrier	1	0	1	0
Bull Mastiff	1	0	1	0
Bulldog	1	0	1	0
Chow Chow	1	0	1	0
Cocker Spaniel	1	1	0	0
Dalmatian	1	0	1	0
German Shorthaired Pointer	1	1	0	0
Golden Retriever	1	0	1	0
Great Dane	1	1	0	0
Irish Setter	1	0	0	1
Irish Wolfhound	1	0	1	0
Karabash	1	1	0	0
Neapolitan Mastiff	1	1	0	0
Northern Inuit Dog	1	0	1	0
Schipperke	1	0	1	0
Weimaraner	1	1	0	0

spayed. Male dogs were significantly more likely to be affected by SADs than female dogs ($P < .0001$). There was no significant age difference between female and male dogs ($P = .9850$).

Overall, the age of onset of clinical signs varied between 2.5 months and 13 years (median 26.75 months, mean 39 months, interquartile range [IQR] 52.125 months). In the group of cervical SADs, the youngest dog developed clinical signs at the age of 2.5 months and the oldest dog at the age of 11.5 years (median 20.25 months, mean 35.6 months). The first signs in dogs with thoracolumbar SADs were noticed between 3 months and 13 years (median 30 months, mean 42.9 months). The median age at time of presentation was 36 months overall (mean 46 months), 30 months median (mean 42.9 months) in the cervical group, and 39 months median (mean 49.4 months) in the thoracolumbar group. There was no significant difference in age at presentation between dogs with a cervical or thoracolumbar SAD ($P = .52$).

Dogs weighed between 1.4 and 84 kg (median 18.3 kg) overall, between 5.7 and 84 kg (median 32 kg) with cervical SADs, and between 1.4 and 57 kg (median 12 kg) with a thoracolumbar SAD. Dogs with a cervical SAD had a significantly higher body weight than dogs with a thoracolumbar SAD ($P < .001$).

Pug dogs (n = 21), Rottweilers (n = 17), and French Bulldogs (n = 13) were the most common breeds. Compared with the respective hospital populations, the highest percentages occurred in Pug dogs, French Bulldogs, and Rottweilers. Of all Pug dogs that had been presented to the Animal Health Trust between 2000 and 2012, 4.5% were diagnosed with a SAD. Of the Pug dog population at the University of Ghent, 3.4% were diagnosed with a SAD and 0% of the Pug dogs at the Royal Veterinary College. The respective percentages for the French Bulldog were 3.4, 1.1, and 0%; and 2.8, 0.7, and 0.16% for the Rottweiler. When compared with the German Shepherd Dog (0.2%, 0.05%, 0%), a common breed in all 3 clinic populations, the overrepresentation of these breeds becomes more apparent.

When comparing the age of the different dog breeds, it became apparent that the Dogue de Bordeaux had a lower age at the time of onset (median 11 months, mean 21.1 months) compared with the study population ($P = .0285$), whereas the Pug dogs were significantly older (median 59 months, mean 51.2 months) than the overall population ($P = .0061$).

Clinical Signs

The most common clinical signs were general proprioceptive ataxia (n = 113, 92.6%) and hypermetria (n = 26, 21.3%). Spinal hyperesthesia was noticed in 23 dogs (18.9%). Only 6 dogs (4.9%) demonstrated obvious paresis. Of these 6 dogs, 50% had a concurrent neurologic disease, such as atlantoaxial instability, vertebral malformation, or a syrinx. Urinary incontinence was present in 4 dogs (3.3%), fecal incontinence was present in 5 dogs (4.1%), and a combination of urinary and fecal incontinence was present in 5 dogs (4.1%). With the exception of 1 dog with fecal incontinence with a dorsal SAD at C1, all dogs with incontinence were diagnosed with a thoracolumbar SAD. Incontinence was significantly more likely to occur in dogs with a thoracolumbar SAD ($P = .0003$) than with a cervical SAD. In 2 dogs, the SAD was considered to be an incidental finding. Both dogs were presented with tetraparesis and diagnosed with a cervical intervertebral disk extrusion. During diagnostic imaging of the whole spinal cord, a SAD in the thoracolumbar region was found, which was thought to be incidental.

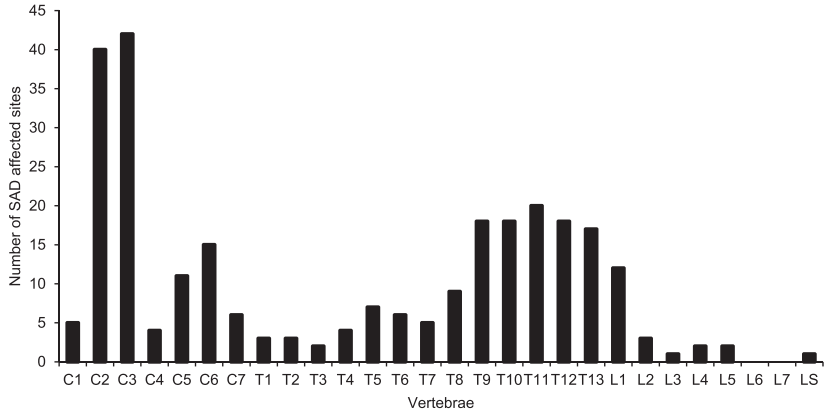

Fig 1. Distribution of SAD over vertebral bodies. In the 122 dogs, SADs were seen over 274 vertebrae. In 25 dogs, the SAD was located only over 1 vertebra, in 86 dogs over 2 vertebral lengths, in 6 dogs over 3, in 3 dogs over 5, and in 3 dogs over more than 8 vertebrae.

Clinical signs were progressive in 91.0% of all dogs. Progression of clinical signs was seen in 61 dogs (95.3%) with a cervical and 50 dogs (86.2%) with a thoracolumbar SAD. In 111 dogs, clinical signs were present for more than 2 weeks before presentation.

Examination of Cerebrospinal Fluid

Cerebrospinal fluid analysis was performed in 65 dogs (53.3%). CSF was collected from the cisterna magna in 19 dogs, 13 of these dogs were diagnosed with a cervical SAD, 1 with multiple, and 5 with thoracolumbar SADs. In 22 dogs, a lumbar CSF collection was performed, 8 of these dogs had a cervical SAD, 13 a thoracolumbar, and 1 multiple. In 1 dog with both a cervical and thoracolumbar SAD, CSF was collected from both sites. The site of collection was unknown in 22 dogs.

TNCC and total protein concentration were within normal limits in 46 dogs (70.8%). An increased protein concentration with an unremarkable TNCC was found in 13 dogs (20%). An increased TNCC with an increased total protein concentration was detected in 6 dogs (9.2%). Cytology in these dogs revealed a monocytic pleocytosis in 4 of these dogs, a lymphocytic pleocytosis in another dog, and was not determined in the remaining dog. For 24 dogs, the CSF samples were submitted for polymerase chain reaction (PCR) for *Toxoplasma gondii*, *Neospora caninum,* and canine distemper virus. All results of PCR analysis for infectious diseases were negative.

Imaging Studies

Myelography demonstrated the typical teardrop-shaped widening of the contrast column at the site of the SAD in 25 dogs. In 11 other dogs, myelography demonstrated a block of the subarachnoid contrast column without filling of the SAD. In these dogs, subsequent MRI demonstrated a focal widening of the subarachnoid space at the location of contrast blockage. In 13 dogs, myelography was suggestive for a SAD and this diagnosis was confirmed using subsequent CT-m. On MRI, all SADs were hyperintense on the T2-weighted sequence and iso- to hypointense on T1-weighted images. Most SADs were hypointense on FLAIR (fluid attenuated inversion recovery) images. The typical teardrop-shaped appearance was also visible on MRI and CT-m.

Previous and Concurrent Neurologic Diseases

In 26 dogs, a concurrent or previously diagnosed spinal cord disorder was detected at the same or an adjacent vertebral level of the SAD. In dogs with a concurrent disease, it cannot be stated whether the signs were attributed to the SAD, to the concurrent spinal disease, or a combination of both. These concurrent disorders included vertebral malformations (n = 11), intervertebral disk extrusion or protrusion (n = 9), atlantoaxial instability (n = 2), vertebral canal stenosis attributable to articular process hypertrophy (n = 1), fibrocartilaginous embolism (n = 1), and myelitis of unknown origin (n = 1). Although not in close proximity to the diagnosed SAD, 2 dogs had a concurrent synovial cyst and 1 dog had a concurrent quadrigeminal cyst.

One dog previously underwent surgery for an intervertebral disk extrusion at the same localization, where it was now diagnosed with a SAD. Two dogs with a cervical SAD had a previous history of steroid-responsive meningitis-arteritis.

Eight of the 13 French Bulldogs (61.5%) and 7 of the 21 Pug dogs (33.3%) had a concurrent disease, whereas only one of the 17 Rottweilers (5.9%) and none of the 5 Dogue de Bordeaux were diagnosed with a concurrent disease. The number of concurrent spinal disorders differed significantly among French Bulldogs, Pug dogs, Rottweilers, and Dogues de Bordeaux ($P = .0001$), with the Pug dog and French Bulldog having a higher number of concurrent spinal disorders and the Rottweiler and the Dogue de Bordeaux having a lower number of concurrent spinal disorders.

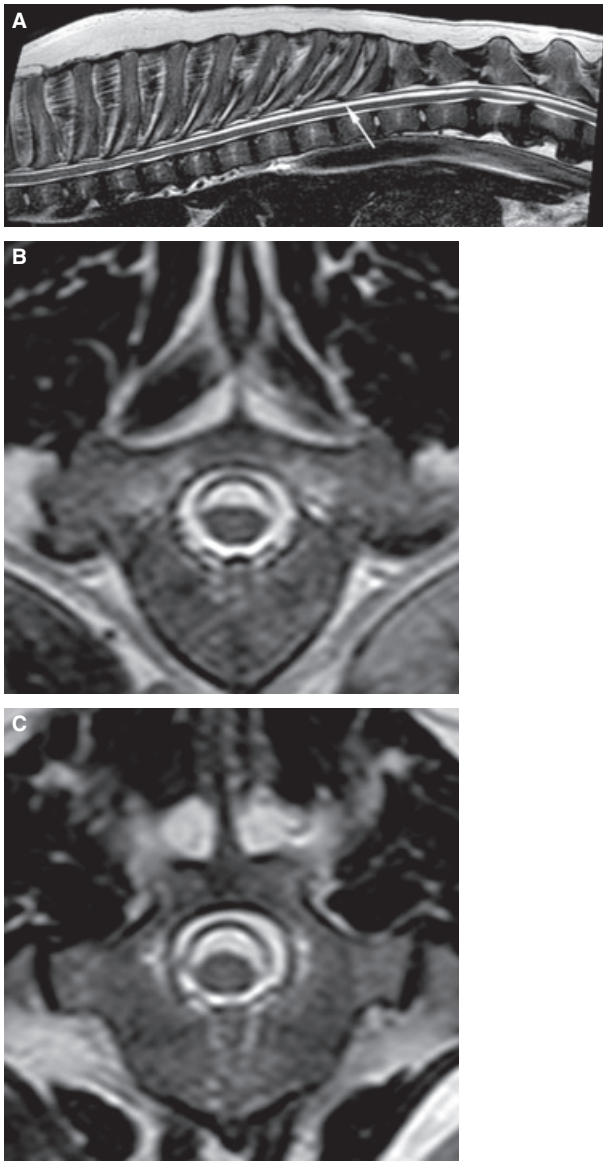

Fig 2. (A) Midsagittal T2-weighted image of the thoracic spine showing a dorsal SAD extending from T1 to L3 in a 74-month-old Northern Inuit with ambulatory tetraparesis and generalized ataxia. The cranial aspect of the SAD is situated 1 vertebral length caudally to a C6-C7 intervertebral disk protrusion. The white arrow indicates the maximal point of enlargment of the dorsal subarachnoid space at the level of T8. (B) Transverse T2-weighted image of the SAD at the level of T8 (indicated by the white arrow in (A)) of the same dog shown in image 2A. (C) Transverse T2-weighted image of the SAD at the level of T12 of the same dog shown in image 2A.

Intervertebral disk extrusion or protrusion occurred concurrently in 4 of the French Bulldogs (30.7%), 3 showed a concurrent vertebral malformation (23.7%), and 1 dog (7.7%) had previous surgery for intervertebral disk extrusion and had also concurrent vertebral malformations. Four Pug dogs (19.0%) had a concurrent intervertebral disk extrusion and 3 (14.3%) had a concurrent vertebral malformation.

Fifty-four dogs with a cervical SAD and 37 dogs with a thoracolumbar SAD did not have any previous or concurrent neurologic diseases. Acquired previous or concurrent diseases, including intervertebral disk extrusion or protrusion, fibrocartilaginous embolism, and steroid-responsive meningo-arteritis, were significantly more common in adult dogs ($P = .032$).

Discussion

This study describes the signalment, clinical findings, and imaging findings in 122 dogs with SADs. Since 1968, only 18 reports of SADs in dogs have been published.[10] These studies involved smaller number of cases, and therefore possible breed or sex predispositions, variability in clinical signs, and occurrence of concurrent spinal disorders were difficult to assess. In this report, we noted a significant overrepresentation of male dogs. Previous authors also reported a trend toward male dogs being more likely to be diagnosed with SADs.[5,7,11,12] In people, two-thirds of SAD patients are male and a possible hormonal influence on SAD formation has been suggested.[13] Studies identified progesterone receptors in the lining of intracranial arachnoid cysts.[14,15] Because CSF volume has been shown to be influenced by hormones,[16] a hormonal impact on SAD formation seems possible and warrants further investigation.

Pug dogs, French Bulldogs, and Rottweilers were the most commonly affected breeds in this study and were overrepresented compared with the respective hospital populations. The overrepresentation of the Rottweiler has been described previously and authors suggested a possible hereditary cause or a biomechanical influence attributable to the heavy head of this dog breed.[5,7,11] In our study, Pug dogs and French Bulldogs were more commonly affected by SADs than the Rottweiler. As in both of these dog breeds concurrent diseases occurred frequently, a predisposition for acquired SADs might be possible. In contrast to this, a spinal cord disorder was only diagnosed in a small minority of Rottweilers and did not occur in the Dogue de Bordeaux. This might support a possible congenital or hereditary etiology of SAD in these breeds.[5,7] Further pedigree evaluation might be warranted to investigate a possible hereditary etiology in these breeds.

One report suggested that younger and heavier dogs might be more prone to cervical SADs.[1] In the study reported here, there was indeed a significant association between weight and localization with heavier dogs being more commonly diagnosed with a cervical SAD. However, age did not have a significant influence on localization of the SAD. As most of the heavier dog breeds in this study are dogs with relatively large heads compared with their body, this might again be explained by the theory previous authors proposed.[5] In accordance with previous reports, the most common cervical localization was C2-3 followed by C5-6.[1,7,17] For thoracolumbar SADs, T13-L1 was the most commonly affected site. Previous studies suggested that this might be caused by a high spinal mobility in this

region.[1,5,7] Again in agreement with previous studies, most SADs resulted in a mid-dorsal localization.[1,7,11] As previously described, many SADs, which completely surrounded the spinal cord, occurred in the Rottweiler, especially in the caudal cervical region.[11] Multilobulated SADs have previously only been described in the Rottweiler,[6] whereas in our study, multilobulated SADs were also observed in other breeds. It has been suggested that the multilobulated SADs represent a specific congenital variant. However, 1 dog in this study had concurrent atlantoaxial instability, suggesting a possible acquired etiology. In agreement with the previous study, all multilobulated SADs occurred in the cervical region.[6]

Because of the middorsal localization of most SADs, the clinical signs of dogs with SADs can differ from dogs with other spinal cord diseases. The most common clinical signs of SADs have been described in previous reports as being ataxia and hypermetria.[1,5,7] These were also the most common clinical signs in this report. As discussed in a previous study, the ataxia is most likely caused by an impairment of the dorsally located ascending proprioceptive pathways and the hypermetria might be explained by the compression of the spinocerebellar tracts, which are located dorsolaterally in the spinal cord.[1] Fecal incontinence was reported in 8.2% of dogs. Upper motor neuron fecal incontinence has been reported to occur more commonly in SAD patients compared with other spinal cord disorders.[18] It has been suggested that this upper motor neuron fecal incontinence is possibly related to dorsal compression of sensory pathways important for conscious defecation.[1,18]

In agreement with a previous study, spinal hyperesthesia was an uncommon feature in dogs with SADs.[1] However, spinal hyperesthesia occurred in 18% of the dogs in this study, which seems slightly higher than in most reports.[1,11] Our findings are comparable to 1 previous study, where 24% of the dogs showed signs of spinal hyperesthesia.[6] Furthermore, some case reports described signs of pain in dogs with SADs.[10,19,20]

Examination of CSF was within normal limits or demonstrated nonspecific abnormalities, such as a mild elevation of TNCC or protein content. These findings have been described in previous studies and are probably linked to the compressive nature of the SAD.[5,6]

The typical imaging findings of SADs have been described previously as being a characteristic teardrop-shaped appearance on myelography or CT-m.[6] On MRI, not all SADs in this study were hypointense on FLAIR (fluid attenuated inversion recovery), suggesting that the fluid is chemically not completely equal to CSF in dogs. A study in people found that the fluid in intracranial arachnoid cysts differs chemically from CSF.[21] This might also be the case in the fluid of canine SADs and warrants further investigation.In previous veterinary and human studies, SADs have been diagnosed concurrently with or after occurrence of compressive spinal cord disorders, spina bifida, inflammatory lesions, or traumatic events, such as previous myelographic studies or spinal surgeries.[1,6,7,19,22–24] In this study, 26 dogs (21.3%) had a previously or concurrently diagnosed spinal disorder. An initial imaging study without evidence of SAD formation followed by a diagnosis of SAD at the same site at a later time was documented in a single dog. These findings would support an association between prior spinal cord injury and subsequent SAD formation for this patient. A possible causative relationship between concurrent neurologic diseases and SAD formation in the other dogs remains speculative. This study is also limited by its retrospective study design, which makes it difficult to decide if the clinical signs in these dogs were attributable to the SAD, the concurrent disease, or a combination of both. Previous or concurrent neurologic diseases in close proximity to the SAD, including intervertebral disk extrusion or protrusion, vertebral malformations, vertebral canal stenosis, fibrocartilaginous embolism, or myelitis of unknown origin, were significantly more common in the French Bulldog compared with the Dogue de Bordeaux or the Rottweiler. This variation might suggest that some breeds are more prone to the acquired SAD variant.

In summary, this study demonstrated that SADs occur most often in the dorsal subarachnoid space, usually leading to clinical signs of ataxia, hypermetria, and upper motor neuron fecal and urinary incontinence without appreciable spinal hyperesthesia. Cervical SADs occur predominantly in heavier dogs, whereas age does not have a significant influence on localization. The most commonly diagnosed dog breeds in this study were Pug dogs, French Bulldogs, and Rottweilers. Male dogs were overrepresented. Twenty-one percent of the dogs in this study had a previous or concurrent spinal disease, including intervertebral disk extrusion or protrusion, vertebral malformations, vertebral canal stenosis caused by articular process hypertrophy, fibrocartilaginous embolism, or myelitis of unknown origin. As these diseases were seen in close proximity to the SAD, they might have an influence on SAD formation.

Footnotes

[a] Omnipaque 240 mg iodine/mL, GE Healthcare, Diegem, Belgium

[b] Lightspeed Qx/i, General Electric Medical Systems, Milwaukee, WI

[c] 0.23 Tesla Airis Mate, Hitachi Medical Corporation, Tokyo, Japan

[d] 1.5 Tesla Intera, Philips Medical Systems, Eindhoven, the Netherlands

[e] 1.5 Tesla Sigma Echospeed System, GE Medical Systems

[f] SAS, SAS Institute, Cary, NC

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Conflict of Interest Declaration: Authors disclose no conflict of interest.

References

1. Skeen TM, Olby NJ, Munana KR, Sharp NJ. Spinal arachnoid cysts in 17 dogs. J Am Anim Hosp Assoc 2003;39:271–282.
2. Cloward RB. Congenital spinal extradural cysts: Case report with review of literature. Ann Surg 1968;168:851–864.
3. Shamir MH, Shahar R, Aizenberg I. Subarachnoid cyst in a cat. J Am Anim Hosp Assoc 1997;33:123–125.
4. Allison N, Moeller RB. Spinal ataxia in a horse caused by an arachnoid diverticulum (cyst). J Vet Diagn Invest 2000;12:279–281.
5. Gnirs K, Ruel Y, Blot S, et al. Spinal subarachnoid cysts in 13 dogs. Vet Radiol Ultrasound 2003;44:402–408.
6. Rylander H, Lipsitz D, Berry WL, et al. Retrospective analysis of spinal arachnoid cysts in 14 dogs. J Vet Intern Med 2002;16:690–696.
7. Jurina K, Grevel V. Spinal arachnoid pseudocysts in 10 Rottweilers. J Small Anim Pract 2004;45:9–15.
8. Ness MG. Spinal arachnoid cysts in two Shih Tzu littermates. Vet Rec 1998;142:515–516.
9. Di Terlizzi R, Platt S. The function, composition and analysis of cerebrospinal fluid in companion animals: Part II-Analysis. Vet J 2009;180:15–32.
10. Gage ED, Hoerlein BF, Bartels JE. Spinal cord compression resulting from a leptomeningeal cyst in the dog. J Am Vet Med Assoc 1968;152:1664–1670.
11. Frykman OF. Spinal arachnoid cyst in four dogs: Diagnosis, surgical treatment and follow-up results. J Small Anim Pract 1999;40:544–549.
12. Chynn KY. Congenital spinal extradural cyst in two siblings. AJR 1967;101:204–215.
13. Dyce J, Heritage ME, Houlton JEF, et al. Canine spinal "arachnoid cysts". J Small Anim Pract 1991;32:433–437.
14. Verhagen A, Go KG, Visser GM, et al. The presence of progesterone receptors in arachnoid granulations and in the lining of arachnoid cysts: Its relevance to expression of progesterone receptors in meningiomas. Br J Neurosurg 1995;9:47–50.
15. Go KG, Blankenstein MA, Vroom TM, et al. Progesterone receptors in arachnoid cysts. An immunocytochemical study in 2 cases. Acta Neurochir 1997;139:349–354.
16. Grant R, Condon B, Lawrence A, et al. Is cranial CSF volume under hormonal influence? An MR study. J Comput Assist Tomogr 1988;12:36–39.
17. Galloway AM, Curtis NC, Sommerlad SF, et al. Correlative imaging findings in seven dogs and one cat with spinal arachnoid cysts. Vet Radiol Ultrasound 1999;40:445–452.
18. Chen AV, Bagley RS, West CL, et al. Fecal incontinence and spinal cord abnormalities in seven dogs. J Am Vet Med Assoc 2005;227:1945–1951.
19. Bagley RS, Silver GM, Seguin B, et al. Scoliosis and associated cystic spinal cord lesion in a dog. J Am Vet Med Assoc 1997;211:573–575.
20. Webb AA. Intradural spinal arachnoid cyst in a dog. Can Vet J 1999;40:588–589.
21. Berle M, Wester KG, Ulvik RJ, et al. Arachnoid cysts do not contain cerebrospinal fluid: A comparative chemical analysis of arachnoid cyst fluid and cerebrospinal fluid in adults. Cerebrospinal Fluid Res 2010;7:8.
22. Kriss TCM, Kriss VMM. Symptomatic spinal intradural arachnoid cyst development after lumbar myelography: Case report and review of the literature. Spine 1997;22:568–572.
23. Andrews BT, Weinstein PR, Rosenblum ML, Barbaro NM. Intradural arachnoid cysts of the spinal canal associated with intramedullary cysts. J Neurosurg 1988;68:544–549.
24. Matthews WB. The neurological complications of ankylosing spondylitis. J Neurol Sci 1968;6:561–573.