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Spinal Arachnoid Diverticula: Outcome in 96 Medically or Surgically Treated Dogs

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Background: Little is reported about the role of medical management in the treatment of spinal arachnoid diverticula (SAD) in dogs.

Objectives: To describe the outcome of 96 dogs treated medically or surgically for SAD.

Animals: Ninety-six dogs with SAD.

Methods: Retrospective case series. Medical records were searched for spinal arachnoid diverticula and all dogs with information on treatment were included. Outcome was assessed with a standardized questionnaire.

Results: Fifty dogs were managed medically and 46 dogs were treated surgically. Dogs that underwent surgery were significantly younger than dogs that received medical management. No other variables, related to clinical presentation, were significantly different between both groups of dogs. The median follow-up time was 16 months (1–90 months) in the medically treated and 23 months (1–94 months) in the surgically treated group. Of the 38 dogs treated surgically with available long-term follow-up, 82% (n = 31) improved, 3% (n = 1) remained stable and 16% (n = 6) deteriorated after surgery. Of the 37 dogs treated medically with available long-term follow-up, 30% (n = 11) improved, 30% (n = 11) remained stable, and 40% (n = 15) deteriorated. Surgical treatment was more often associated with clinical improvement compared to medical management (P = .0002).

Conclusions and Clinical Importance: The results of this study suggest that surgical treatment might be superior to medical treatment in the management of SAD in dogs. Further studies with standardized patient care are warranted.

Key words: Arachnoid cyst; Spinal cord; subarachnoid cyst.

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Clinical signs of 14 dogs have been described in a previous publication: De Decker S. et al. Spinal arachnoid cysts in dogs: a retrospective study of 14 cases. Vlaams Diergeneeskundig Tijdschrift 2006; 75, 153–164. (Article in Dutch)

Signalment and clinical signs of these dogs have been described in a previous publication: Mauler A., De Decker S., De Risio L., Volk H.A., Dennis R., Gielen I., Van der Vekens E., Goethals K., Van Ham L. Signalment, clinical presentation and diagnostic findings in 122 dogs with spinal arachnoid diverticula. JVIM 2014 Jan–Feb;28 (1):175–81.

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Abbreviations:

CSF	cerebrospinal fluid
MRI	magnetic resonance imaging
SAD	spinal arachnoid diverticulum

Surgical treatment of dogs with compressive myelopathy caused by a spinal arachnoid diverticulum (SAD) has been described.[1–7] Several surgical techniques have the aim of spinal cord decompression and simultaneous prevention of SAD recurrence.[6,7] Little is reported about the role of medical management for SAD in dogs. Because the underlying etiology of SAD formation in dogs is yet unknown, there is no causative medical treatment available. Medical treatment can consist of combinations of anti-inflammatory drugs, medications that decrease the amount of cerebrospinal fluid (CSF) production, and supportive physical treatment. To date, the outcome after medical management for SAD has been described for 5 dogs.[1,8–10] In a previous study, we described the signalment, clinical and imaging findings of 122 dogs with SADs, but did not provide information regarding treatment options and associated outcome.[11] The primary aim of the present study was therefore to describe the results of medical and surgical treatment of SAD in a larger population of dogs. Our secondary aims were to compare the signalment, clinical presentation and outcome of dogs that underwent medical or surgical treatment for SAD. We hypothesized that the signalment and clinical presentation of dogs undergoing medical or surgical treatment for SAD would not differ and that although medical management of SAD would be effective for a proportion of studied dogs, it would be associated with a less successful outcome compared to surgical treatment.

Materials and Methods

This study was in accordance with the local ethical and welfare committee guidelines of all participating centers.

Dogs

Medical records and imaging findings of dogs with a diagnosis of SAD between January 2000 and March 2012 were retrospectively reviewed. The dogs were diagnosed with SAD at the Centre of Small Animal Studies, Animal Health Trust, England (n = 41); Faculty of Veterinary Medicine, Ghent University, Belgium (n = 40), and the Royal Veterinary College, University of London, England (n = 15). Only dogs with complete medical records, including information regarding age at diagnosis, sex, bodyweight, duration of clinical signs, presenting clinical signs, previous or concurrent neurological diseases, and treatment were included. For the purpose of this study, dog breeds with an adult weight of more than 20 kg were considered large. Diagnosis was achieved by magnetic resonance imaging (MRI) in 56 dogs, by CT-myelography in 10 dogs, by myelography in 24 dogs and 6 dogs underwent both myelography and MRI. The specific imaging details have been reported previously.[9]

Data Collection Regarding Treatment and Follow-Up

Medical records were reviewed concerning information on treatment, complications, and neurological status at time of discharge and short-term follow-up visit. It was assessed, if there was a difference between medically versus surgically treated dogs, regarding their sex, age at onset of signs, breed, bodyweight, duration of clinical signs, the localization of the SAD, the presenting clinical signs, the progression of signs, and the presence of a concurrent neurologic disorder.

Medical treatment was defined as any treatment, which did not involve surgical intervention. This included dogs receiving medications to decrease the amount of CSF and analgesics. Some of these dogs also underwent physical rehabilitation.

Surgically treated dogs underwent either a durotomy, a durectomy, or a marsupialization to drain the accumulated fluid from the subarachnoid space. All surgical procedures were performed under general anesthesia and dogs received perioperative pain management. Surgeries were performed by a board-certified neurologist or a PhD in orthopedic surgery with 25 years of neurosurgical experience.

If available, the results of a short-term follow-up visit between 3–6 weeks (median 4 weeks) after surgery were reviewed.

Information regarding long-term follow-up was obtained using a standardized questionnaire (Appendix S1). Either the referring veterinarian was contacted by email or telephone, or both the veterinary surgeon and owner were contacted by telephone.

Outcome was categorized into 3 different subgroups: improved, stable and deteriorated. Dogs with an initial improvement and subsequent deterioration were considered as deteriorated. This assessment was based on the questionnaire provided by the owner or referring veterinarian.

Statistical Analysis

A logistic regression model was used to assess, if there were differences between the medically and surgically treated dogs regarding sex, bodyweight, age at onset, age at diagnosis, breed, duration of signs, localization of the SAD, clinical signs, progression of signs, and the presence of a concurrent neurologic disorder. These same variables were also compared between the improved, stable and deteriorated dogs. Then a logistic regression model was used to compare the outcome of the medically versus the surgically treated dogs. Commercially available software was used for all analyses and significance was defined as $P < .05$[a].

Results

Signalment and Clinical Presentation

Ninety-six dogs were included. Fifty dogs (52%) were treated medically and 46 (48%) were treated surgically.

Medically Treated Dogs. In the medically treated group, 38 dogs were male (10 neutered) and 12 dogs (6 neutered) were female. The median age at onset of clinical signs was 45.3 months (mean: 46.2 months) with a range between 3.5 and 156 months. The median weight was 16.4 kg (mean: 24.1 kg) ranging between 6.1 and 61.5 kg. Nineteen different breeds were included in this group. The most common breed was the pug (n = 11), followed by the French bulldog (n = 10) and the Rottweiler (n = 7). Clinical signs had been present for 3 days to 41 months, with a median of 3 months (mean: 7.3 months). Signs were progressive in 41 dogs (82%) and non-progressive in 9 dogs (18%). All 50 dogs showed ataxia and 3 dogs additional paresis. Five dogs had fecal incontinence, 2 urinary incontinence and in 1 dog fecal and urinary incontinence was present. Nine of the dogs in this group showed signs of discomfort upon palpation of the vertebral column. Nine dogs (18%) had a concurrent neurologic disorder. Two dogs had a concurrent intervertebral disk herniation, 3 dogs had vertebral malformations, 2 dogs had other cystic structures (1 synovial cyst and 1 quadrigeminal cyst), 1 dog had concurrent myelitis and 1 dog a fibrocartilaginous embolism. All concurrent disorders, which affected the vertebral column, occurred at the same site as the SAD or in the vertebral segment adjacent to it. Cervical SADs occurred in 22 dogs (44%), 27 dogs (54%) had a thoracolumbar SAD, and 1 dog had multiple SADs in the cervical region.

Surgically Treated Dogs. This group consisted of 37 male (13 neutered) and 9 female (4 neutered) dogs. The youngest dog was 3 months old at time of onset of clinical signs and the oldest 124 months. The median age was 18 months (mean: 31.3 months). The median bodyweight was 17.4 kg (mean: 20.4 kg), ranging from 1.4 to 49 kg. The pug (n = 6), Rottweiler (n = 4) and West Highland White Terrier (n = 4) were the most common breeds in this group. The duration of clinical signs ranged from 1 day to 24 months, with a median of 5 months (mean: 2 months). Signs were progressive in 43 dogs (93%) and nonprogressive in 3 dogs (7%). Ataxia was present in 45 dogs with additional paresis in 3 dogs, additional urinary incontinence in 1 dog and additional fecal and urinary incontinence in 2 dogs. One dog showed only signs of cervical hyperesthesia without any neurologic deficits. Signs of discomfort upon palpation of the vertebral column were noted in 10 dogs. Ten dogs (22%) receiving surgical treatment were diagnosed with a concurrent neurologic disorder. The most common concurrent disorder diagnosed at time of diagnosis of the SAD, were vertebral

malformations, which occurred in 7 dogs, followed by intervertebral disk herniations in 2 dogs. These occurred at the same site as the SAD or in the vertebral segment adjacent to it. One dog had a previous history of a steroid-responsive meningitis-arteritis. Twenty-four (52%) of the surgically treated dogs had a cervical SAD, 20 dogs (43%) a thoracolumbar SAD and 2 dogs had multiple SADs in the thoracolumbar area.

Comparison of Signalment and Clinical Presentation in Medically and Surgically Treated Dogs. When comparing the differences in clinical presentation between medically and surgically treated dogs, only the age at onset of clinical signs was significantly different, with surgically treated dogs being younger than dogs that underwent medical management ($P = .028$).

Treatment

Medically Treated Dogs. Medical treatment consisted of administration of prednisone in 44 dogs. When dosages were available from the medical records, they ranged from 0.3 to 1 mg/kg/d (median 0.65 mg/kg/d). Physical treatment without any further medical treatment was performed in 3 dogs and carprofen at 2 mg/kg twice daily was used in 2 dogs and gabapentin at 10 mg/kg 3 times daily in 1 dog. Three dogs underwent physical rehabilitation in addition to their medical treatment.

Surgically Treated Dogs. The surgical treated dogs underwent one of several surgical techniques, which were chosen based on the surgeons' preference. Twenty-eight dogs underwent a durectomy with removal of the arachnoid adhesions, 15 a durotomy, and 3 a marsupialization. The mean duration of hospitalization was 5.7 days with a median of 5 days. One dog suffered from a major perioperative complication. This dog underwent a durectomy at C2-3 and experienced postoperative hypoventilation 12 hours after surgery, which made ventilatory support necessary. Imaging showed formation of a compressive hematoma, which was surgically removed. The dog required ventilation for 7 days after revision surgery and was discharged 9 days after the second surgery. Minor surgical complications included severe blood loss, requiring a blood transfusion in 1 dog, early postoperative transient neurologic deterioration in 22 dogs and urinary tract infections reported in 4 dogs.

Outcome

Medically Treated Dogs. Follow-up information was available for 37 of the 50 dogs (74%) in this group. A median follow-up of 16 months (range 1–90 months) was available. At the time of data collection 15 dogs (40%) were still alive, 16 dogs (43%) were euthanized as a consequence of the SAD and 6 dogs (17%) died or were euthanized due to reasons unrelated to SAD. Dogs, which were euthanized as a consequence of the SAD had a mean survival of 15.2 months after diagnosis (median 14 months). Seven of these dogs were diagnosed with a cervical and 9 dogs with a thoracolumbar SAD.

Eleven of the 37 medically treated dogs improved (30%), 11 remained stable (30%) and 15 deteriorated (40%). Eight of 31 dogs (26%) treated with prednisone with known outcome improved, 9 remained stable (29%) and 14 deteriorated (45%).

Surgically Treated Dogs. Follow-up information was available for 38 of the 46 surgically treated dogs (83%). Twenty-five of these dogs (66%) were alive at the time of data collection, 5 dogs (13%) were euthanized due to SAD and 4 dogs (11%) had died or had been euthanized due to an unrelated cause. Euthanasia due to the SAD was performed mean 12 months after surgery (median 13 months). Three of the euthanized dogs underwent surgery for a thoracolumbar SAD and 2 had a cervical SAD.

Thirty-one dogs improved (82%), 1 remained stable (3%) and 6 dogs deteriorated (15%).

One dog made an initial improvement and deteriorated 9 months after surgery. Magnetic resonance imaging was performed and revealed a laminectomy membrane formation, which was surgically removed and lead to a good recovery confirmed by telephone interview with the owners 24 months after the second surgery. The dog was ambulatory with mild residual ataxia at this time.

Comparison of Follow-Up Time and Outcome in Medically and Surgically Treated Dogs. A median follow-up time of 16 months (range 1–90 months) was available for the medically treated dogs and 23 months of follow-up for the surgically treated cases. This difference was not significant ($P = 0.64$).

In the medically treated group significantly more dogs deteriorated. Likewise, there were significantly more surgically treated dogs in the improved group ($P = .0002$).

There was no significant difference in outcome for cervical versus thoracolumbar SADs ($P = .65$) or large versus small dog breeds ($P = .18$).

Discussion

The present study retrospectively described and compared different treatment options for dogs with SADs. Since 1968, 20 reports about treatment of SADs in dogs are published, with 5 dogs medically treated.[1,8–10,12] This study reported the clinical presentation of 50 and outcome of 37 dogs treated medically for SADs.

To compare medically and surgically treated dogs as objectively as possible, signalment, clinical signs, progression of signs, and the presence of concurrent neurologic disorders were assessed and compared between both treatment groups. The medically and surgically treated groups differed only significantly in the age at onset of clinical signs with younger dogs undergoing surgical treatment more often. This difference might be explained by the fact that owners are more willing to pursue surgical, cost-intensive treatment in younger dogs.

In both treatment groups, the localization of the SADs was almost evenly distributed between cervical

and thoracolumbar sites. There was no difference in outcome depending on the localization.

Prednisone was administered to 44 dogs in the medically treated group and this treatment for SADs has been described previously.[1,8] Prednisone reportedly decreases the production of CSF and increases its absorption in addition to its anti-inflammatory properties and effects on vasogenic edema. However, other studies using prednisolone did not demonstrate a decrease in CSF pressure.[13] Despite the lack of clear evidence, prednisone is used widely to treat dogs with fluid accumulation in the subarachnoid space. The efficacy of this treatment in dogs has not been clinically studied and is based on empiric observations. Of the dogs treated with prednisone, only 26% improved with a median follow-up of 24 months. Of the 2 previously reported cases, which were treated medically, 1 improved and had a follow-up of 1 year.[8] The other dog remained stable over a period of 21 months.[1]

Other medications used in this study for medical treatment included nonsteroidal anti-inflammatory medications. There has been no report of their use in dogs with SADs. Their use in SADs might be warranted in dogs with spinal hyperesthesia, as they have an analgesic effect. Some dogs were treated with gabapentin, which has been recommended for the treatment of neuropathic pain in dogs with syringohydromyelia.[14] In the present study, only 1 dog was treated with gabapentin, which makes conclusions about its efficacy in SAD treatment impossible.

Different surgical techniques were used in this study depending on surgeon's preference. Durotomy and durectomy were most commonly performed. These techniques have been described previously in the treatment of SADs.[1,4] Durectomy and marsupialization have been suggested to represent better treatment options because of their increased likelihood of permanent drainage of the subarachnoid space.[1,7]

Our finding that a durotomy might be a valid treatment option is difficult to compare to findings in human patients. Subarachnoid cysts in people are usually better demarcated and can often be completely removed.[15,16] The space occupying lesion in the subarachnoid space in people causes compression of the spinal cord, which leads to neurologic signs. In cases where the cysts are more widespread and are non resectable, fenestration of the cyst has been described.[17–19] These techniques are comparable to durotomies or durectomies. However, durectomies or marsupializations without closure of the subarachnoid space are uncommonly performed in people due to the occurrence of orthostatic headaches associated with uncontrolled CSF leakage and the higher rate of recurrence of the cyst.[20,21] Therefore, surgical management in people aims toward cyst removal and seems to achieve better outcomes with subsequent closure of the subarachnoid space.[20] Other proposed surgical techniques in human medicine include shunt placement to permanently drain the intraarachnoid diverticulum in a controlled manner.[20] In dogs, the occurrence of orthostatic headaches is probably less likely due to the quadrupedal gait. However, there might be a number of undiagnosed orthostatic headaches in dogs due to the difficulty in diagnosing headaches in animals.[22]

When comparing the outcome between the medically and surgically treated dogs, significantly more dogs improved in the surgically treated group. This supports previous reports, which suggest surgery as the treatment of choice in canine SAD.[1,6,7] Spinal arachnoid diverticula cause slowly progressive clinical signs, most likely by slow growth of the diverticulum. Medications such as prednisone work through decreasing possible secondary inflammation and edema, although this does not lead to a long-lasting relief of clinical signs. The influence of prednisolone on CSF production might decrease the accumulation of fluid in the SAD. However, in this study, corticosteroid treatment has an inferior outcome compared to surgical treatment.

Although the results of this study indicate that surgical intervention might be superior to medical management for dogs with SAD, surgery is more invasive and pet owners might be hesitant to pursue this route due to financial concerns and concerns about postoperative deterioration and care.

Recurrence of SAD formation has been reported as an important long-term complication after surgery. However, in our study, only one of the dogs had a relapse and was diagnosed with a laminectomy membrane as the cause of deterioration. It is therefore possible that not all dogs with recurrence of clinical signs have recurrence of SAD formation and that the recognition of other complications might be underestimated. The outcome in this study was purely based on clinical signs and the long-term follow-up was subjectively assessed by the owner or the referring veterinarian.

This study was obviously limited by its retrospective nature, which limited standardization of assessment and veterinary care. The outcome was assessed using a questionnaire, but no follow-up visits were conducted. Multiple institutions from different countries were involved, which could have affected the choice between medical and surgical management. Most dogs presented to referral centers in the UK have medical insurance, while this practice is not common in Belgium. It is therefore possible, that financial reasons were an unrecognized confounding variable in this population of dogs. It is also possible, that other factors, such as the presence of incontinence or the severity of clinical signs, biased the decision for medical or surgical treatment.

Although several of these limitations could have been avoided by a prospective study design, retrospective studies have the advantage of including a larger number of animals, which is of major importance in a relatively rare disease such as SAD.

In summary, this study compared a relatively large number of medically and surgically treated dogs with SAD. It supports the previous suggestion that surgical treatment is a good treatment option for dogs with SAD. Due to the retrospective nature of this study, it is not possible to know, whether surgery is truly the superior option and which surgical technique is preferable. These questions warrant further prospective investigation.

Footnote

[a] SAS, SAS Institute, Cary, NC

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

Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

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Supporting Information

Additional Supporting Information may be found online in the supporting information tab for this article:

Appendix S1. Standardized questionnaire to follow-up spinal arachnoid diverticula.