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Short- and long-term outcome and magnetic resonance imaging findings after surgical treatment of thoracolumbar spinal arachnoid diverticula in 25 Pugs

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Abstract

Background: There is a successful outcome after surgical management of spinal arachnoid diverticula (SAD) in up to 82% of cases.

Hypothesis/Objectives: We hypothesized that Pugs have favorable short-term and poor long-term prognosis after surgical treatment of thoracolumbar SAD. The aim of the present investigation was to describe clinical findings, short- and long-term outcomes, and follow-up magnetic resonance imaging (MRI) findings in Pugs with thoracolumbar SAD.

Animals: Twenty-five client owned Pugs with 12-month follow-up information after surgical treatment of thoracolumbar SAD.

Methods: Multicenter retrospective case series. All medical records were searched for Pugs diagnosed with SAD. Data regarding signalment, history, surgical procedure, outcome, histopathology, and follow-up MRI results were extracted.

Results: Mean age at presentation was 7.32 (range 2-11) years, 80% were males. Short-term outcome was available in 25 dogs, and improvement was confirmed in 80% of dogs. Long-term outcome was available in 21 dogs, and deterioration was confirmed in 86% of cases, with late-onset recurrence of clinical signs after initial postsurgical improvement affecting 85% of Pugs. A moderate correlation (r = 0.50) was found between duration of clinical signs and outcome. In 8 dogs with deteriorating clinical signs, follow-up MRI revealed regrowth of the SAD in 2 cases, new SAD formation in 2 cases, and intramedullary T2W hyperintensity/syringomyelia in 6 cases.

Conclusions and Clinical Importance: This study suggests that Pugs with thoracolumbar SAD do not have a favorable long-term prognosis after surgical treatment for reasons yet to be determined.

KEYWORDS

Pugs, SAD, spinal cyst, surgical treatment of SAD

Abbreviations: CT, computed tomography; H&E, hematoxylin and eosin; IVDD, intervertebral disc disease; MRI, magnetic resonance imaging; SAD, spinal arachnoid diverticula; TSCIS, Texas spinal cord injury score.

1 | INTRODUCTION

Spinal arachnoid diverticula (SAD) is a fluid accumulation in the subarachnoid space causing signs of neurological dysfunction such as paresis, spinal ataxia, urinary and fecal incontinence because of a secondary compressive myelopathy.[1–7] It occurs in humans and companion animals, including dogs and cats.[2,7–9] In dogs, SAD occurs in cervical and thoracolumbar areas.[2,3,7,10,11] Common breeds affected by SAD in the cervical area include Pugs, Rottweilers, and Labrador Retrievers,[3,7,10] whereas in the thoracolumbar area Pugs, French bulldogs, and West Highland white terriers seem to be more frequently affected.[3,4,6,7] Large and giant breed dogs are predisposed to have cervical SAD and middle to small breed dogs are predisposed to develop thoracolumbar SAD.[2,7]

Medical and surgical treatments of SAD are described in dogs.[3–6,12–14] Surgical treatment has a better outcome in comparison to medical management.[12] Surgical treatment outcome success of cervical and thoracolumbar SADs does not differ significantly.[12] Success rates of surgical management of thoracolumbar SADs in various breeds are up to 82%.[3,12] In Pugs, mostly surgical management of SAD with various results is reported.[3–6,15,16] Successful outcome after surgical treatment of SAD reaches up to 75% in Pugs.[3–6,15] Reported follow-up period ranges from 2 months to a few years, with a follow-up of 6 months or longer being available only in 6 Pugs.[3–6,15,16] A follow-up of longer than 12 months in Pugs after surgical management of SAD is described in 4 Pugs with recurrence of signs of neurological dysfunction occurring in 3 of them in the time period between 19 and 29 months postsurgically;[6,15,16] therefore, we made an attempt to define the long-term prognosis in this breed after surgical treatment of SAD.

Aims of the current study included the examination of short- and long-term outcome in Pugs after surgical management of thoracolumbar SADs, description of the clinical, initial, and follow-up diagnostic imaging and histopathological findings, and investigation of factors associated with outcome in Pugs with confirmed SAD. We hypothesized that Pugs have short-term improvement and long-term deterioration after surgical management of thoracolumbar SAD and that recurrence of SAD was the main reason for long-term deterioration in this breed with intraoperative leptomeningeal adhesion dissection and marsupialization being the factors associated with better outcome.

2 | MATERIALS AND METHODS

The current study is in accordance with local ethical and welfare committee guidelines of all involved veterinary centers.

The study was performed at 3 veterinary centers—Small Animal Clinic of the University of Zurich (Vetsuisse Faculty, University of Zurich, Zurich, Switzerland), Dick White Referrals Hospital (Six Mile Bottom, Cambridgeshire, UK), and the Referral Veterinary Hospital Aisti (Vantaa, Finland). Client owned dogs were included. Veterinary facilities databases were reviewed retrospectively from the time interval from 2006 to 2016. Inclusion criteria were Pugs with surgically confirmed and surgically treated SAD in the thoracolumbar area, available clinical data, including breed, age at presentation, clinical signs at presentation, results of neurological examination, duration of clinical signs, diagnostic imaging results (magnetic resonance imaging [MRI] or myelography), description of surgical procedure, and outcome for at least 12 months after surgical treatment of SAD. If the neurological status deteriorated or euthanasia was performed because of signs of neurological dysfunction related to SAD earlier than 12 months after the surgery, dogs were still included in the study. Information about the histopathological examination of the excised SAD or postmortem histopathological examination results were included, if available.

Dogs were anesthetized and euthanized according to ethical principles using anesthetic medications. Magnetic resonance imaging scans and surgeries were performed under general anesthesia with assisted or spontaneous ventilation; dogs were positioned in dorsal recumbency for the MRI scans and sternal recumbency for the surgical procedures.

In 14 cases, radiological diagnosis was made at the referral small animal hospital Aisti using a low-field 0.2 T MRI scanner (Vet-MR, Esaote S.p.A, Italy) or 0.3 T MRI scanner (Vet-MR Grande, Esaote S.p.A). In 5 cases, diagnosis was made at the veterinary referral center Dick White Referrals using a low-field 0.4 T MRI scanner (Hitachi Aperto, Hitachi Medical Corporation, UK), and in 6 dogs, diagnosis was made at the Small Animal Clinic of the University of Zurich using a high-field 3 T MRI scanner (Philips Ingenia, Philips AG, Switzerland). The same MRI scanners were used for follow-up MRI examinations if dogs were presented with recurrence of signs of neurological dysfunction. T2W sagittal and transverse plane images were acquired in all study dogs. Standard MRI protocols were T2W, T1W sagittal, and T2W transverse plane images at Aisti animal hospital; T2W sagittal and T1W and T2W transverse plane images at Dick White Referrals animal hospital; and T2W sagittal and transverse plane images, in addition to short tau inversion recovery dorsal plane images at the animal hospital of University of Zurich. In some individuals, additional MRI sequences were acquired. Images were reviewed, and radiological as well as clinical diagnosis was made by board certified neurologists (S. Cizinauskas, F. Steffen, and G.B. Cherubini). It was noted if hemivertebrae occurred in proximity to the site of the SAD or intervertebral disc disease (IVDD) and spondylosis deformans were found close to the SAD. Associated hemivertebrae, IVDD, and spondylosis deformans were considered relevant if they occurred at the level of SAD or 1 vertebral body or intervertebral disc space cranially and caudally to it.

Surgeries were performed by ECVN diplomates (S. Cizinauskas, F. Steffen, and G.B. Cherubini) and experienced neurosurgeons with at least 10 year of surgical experience (M. Rakauskas, J. Jeserevics). Surgical procedures included standard hemilaminectomy (16 cases) and dorsal laminectomy (9 cases) approaches followed by identification of externally bulging dura mater and excision of the abnormal part of the dura mater using beaver blades or No. 11 surgical blades. Durectomy was performed with an attempt to remove the diseased dura mater until the edge of the adjacent normal dura mater. The adhesions between the arachnoid and the pia mater (if they were present) were either gently dissected from each other or it was left untouched with only the bulging part of the dura mater removed. In 5 of 25 cases, the dura mater was marsupialized with 2 stitches using

5/0 nonabsorbable suture material. Edges of the excised dura mater were sutured to the remnants of the articular capsule dorsally and ventrally. After surgery, the dogs were either hospitalized or sent home with standard anti-inflammatory, analgesic medications, or both, according to the protocols of the different institutions.

Histopathological examination of the excised dura mater was performed in 12 cases by a board certified pathologist (M. Anttila) at the PATOVET (Animal Pathology Laboratory, Helsinki, Finland). Dura mater samples were fixed in neutral-buffered 4% formalin solution, routinely processed and stained with hematoxylin and eosin (H&E).

Postmortem examination was performed by a board certified pathologist (A. Oevermann) in the Institute of Animal Pathology at the University of Bern (Vetsuisse Faculty, Department of Infectious Diseases and Pathobiology, University of Bern, Bern Switzerland) in 1 study case. Spinal cord and adjacent dura mater were fixed in neutral-buffered 4% formalin solution, routinely processed and stained with H&E.

Short-term outcome was defined as the neurological status at 6 months after surgical treatment of SAD, and long-term outcome was defined to be neurological function at 12 months or later after the surgical treatment of SAD. Short and long-term outcomes were evaluated either during control examination, utilizing Texas spinal cord injury score (TSCIS) for each hind limb separately,[17] at the veterinary clinic where the surgery of SAD was performed or outcome evaluation was based on a custom-designed telephone questionnaire (Supplemental Appendix 1). Outcome was considered improved if dogs showed improvement of motor function of the pelvic limbs, improved defecation, urination, or combination of the above mentioned. Outcome was considered deteriorated if dogs had decline of motor function of the caudal limbs, developed fecal, urinary incontinence, or combination of the above mentioned. If neurological status of a dog was unchanged, the outcome was defined as stable.

To evaluate the correlation between duration of clinical signs before presentation and outcome, Pearson's correlation coefficient was calculated. Student *t* test was performed to evaluate the association among perioperative (≥2 months) anti-inflammatory drug usage, intraoperative marsupialization, and leptomeningeal adhesion dissection and outcome. Association was considered significant if $P < .05$. Outcome was defined as the time period between surgery of SAD and recurrence of signs of neurological dysfunction. If dogs were showing stable signs of neurological dysfunction or improving, they were excluded from this analysis. Statistical analyses were performed using Medcalc statistical software (MedCalc software bvba, Belgium).

Follow-up MRI was available in 8 dogs from 7 to 48 months after surgical treatment of the SAD: 3 of them were performed at veterinary referral hospital Aisti, 1 at veterinary referral hospital Dick White Referrals using low-field MRI, and 4 at the small animal clinic of the University of Zurich using high-field MRI.

3 | RESULTS

Twenty-five Pugs were included in the study. Fourteen dogs were presented to the veterinary hospital Aisti (Finland), 5 to the referral hospital Dick White Referrals (UK), and 6 dogs to the Small Animal Veterinary Clinic of the University of Zurich (Switzerland). Age of onset of signs of neurological dysfunction ranged from 2 to 11 years (mean 7.32 years, SD ± 1.86). Five dogs were females and 20 were males. The male : female ratio was 4 : 1. Twenty-four dogs were presented with chronic clinical signs of spinal ataxia and ambulatory paraparesis in the hind limbs, 2 of which had concomitant urinary and fecal incontinence. One dog had a chronic spinal ataxia and ambulatory paraparesis, but had acute worsening of signs of neurological dysfunction a week before presentation and was presented at the hospital with nonambulatory paraparesis. Texas spinal cord injury score for right/left hind limbs at presentation ranged from 5/5 to 8/8. Duration of clinical signs at presentation ranged from 1 to 36 months with median duration of 3 months. No study dog was treated with nonsteroidal anti-inflammatory drugs for longer period than 14 days. Steroidal anti-inflammatory drugs (prednisolone) in the perioperative period for 2 months or longer were used in 5 cases. In 8 cases, prednisolone therapy was started from 8 to 30 months post-surgery with improvement (1/8), without improvement (3/8), or with unknown therapy results (4/8). Perioperative prednisolone use was not associated with better outcome ($P = .14$).

Spinal arachnoid diverticula were diagnosed in all 25 study Pugs by means of MRI examination. In the current study where only thoracolumbar SADs were investigated, it was located in the thoracic area in all dogs, ranging from T6 to L1 level. Concurrent MRI findings included IVDD in associated vertebras in 14 cases (58%, 14/24), spondylosis deformans in 5 cases (21%, 5/24), and vertebral malformations—hemivertebrae—in 2 cases (8%, 2/24). In 1 dog, the information about concomitant vertebral malformations was not available. Vertebral articular joint hypoplasia, aplasia, or both, was detected in 6 cases of dogs which were scanned with high-field MRI scanners or which were additionally examined using radiographs of the vertebral column. It was not possible to reliably evaluate this type of malformation using low-field MRI scanners because of wider slice thickness and lack of image definition.

There was TSCIS score available at different follow-up time points in 22 cases, and the score ranged from 2/2 to 9/9. Short-term outcome after the surgery was available in all dogs. Improvement of motor function, defecation, urination, or combination of the above mentioned, was recorded in 80% (20/25) of the cases. Unchanged neurological status after surgical treatment of SAD was recorded in 1 case, 4 dogs showed deterioration, with 2 of them euthanized 3 weeks and 6 months after SAD surgical treatment. The other 2 surviving dogs had deterioration immediately and 3 months after the surgery with 1 of them having slowly progressing deterioration up to the 18 months time point post-surgery and the other 1 lost to follow-up during deteriorating neurological state.

Long-term outcome at 12 months or later time point was available in 21 dogs. Further improvement in comparison to neurological status 6 months after surgery was recorded in 5% (1/21) of cases. Two of 21 (10%) dogs showed improvement at the 6 months time point after surgical management and stayed in a stable neurological status at 12 months or later follow-up time point. Deterioration was detected in

86% (18/21) of Pugs. Other 4 Pugs were not available for the long-term follow-up because 3 of them were euthanized 3 weeks, 6 months, and 11 months after the surgery, and 1 dog with worsening of signs of neurological dysfunction 3 months after the surgery was lost to follow-up. Initial improvement followed by recurrence of clinical signs was recorded in 85% (17/20) cases. Moderate correlation was found between the duration of clinical signs before presentation and outcome measures (r = 0.50). Intraoperative marsupialization and dissection of leptomeningeal adhesions were not associated with the outcome (P = .81 and P = .56, respectively).

Histopathological examination of excised dura mater was available in 12 cases. In 6 cases the dura mater was fibrotic, in 5 cases it was infiltrated with inflammatory cells (Figure 1), in 4 cases normal or thickened dura mater was found, and in 1 case signs of hemorrhage were confirmed. In 1 Pug, postmortem examination was performed and dural fibrosis with leptomeningeal adhesions were confirmed with white matter changes including gliosis, axonal degeneration, and syringohydromyelia.

In dogs with recurrence of signs of neurological dysfunction, follow-up MRI examinations revealed recurrence of the SAD in 2 cases, formation of new SAD in other 2 cases, and advanced intramedullary T2W hyperintensity, syringomyelia, or both in 6 cases (Figures 2–4).

4 | DISCUSSION

In the current study we found that 80% of Pugs show improvement of signs of neurological dysfunction within the first 6 months after surgical treatment of SAD. We believe that short-term improvement prevalence is even higher as 1 of the dogs with short-term deterioration acutely deteriorated owing to suspected traumatic event. However, in 85% of the dogs with initial improvement, deterioration of clinical signs was observed in the time period between 7 and 48 months after the surgical treatment. Therefore, long-term outcome rate in the current study was much lower compared to studies which included various dog breeds with SAD. In studies describing outcome after surgical treatment of SAD including all dog breeds, there was improvement in 66% and 82% of the cases with median follow-up of 23 months (15 and 38 surgically managed cases included).[3,12] In another study with 13 dogs suffering from SAD, there was postsurgical improvement in 66% of cases with follow-up period ranging from 6 to 30 months.[14] Recurrence of signs of neurological dysfunction in dogs with initial improvement occurred in 25% of the cases in the previous study which included 15 surgical cases of SAD,[3] and only a few cases of recurrence are reported in 2 studies with 38 and 14 surgical cases involved.[12,18] In the current study, short-term outcome rates are similar to the follow-up results in all dog breeds. In contrast, long-term outcome differs substantially compared to the published outcome in a population of various breed dogs.

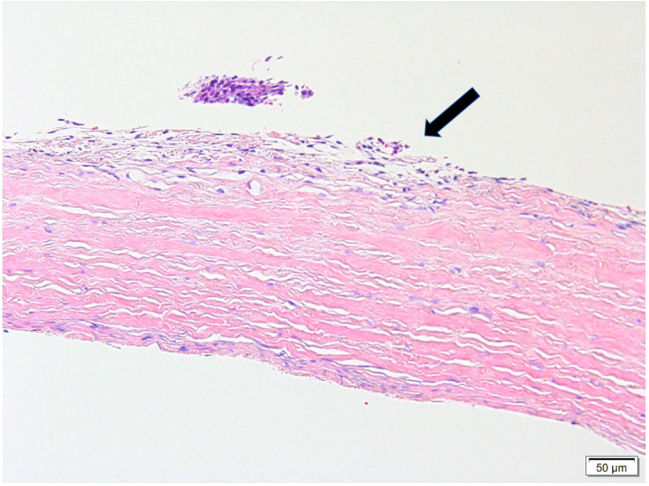

FIGURE 1 Pug, 7 years old, male, suffering from SAD at the T11 vertebral level. Histological section of the diverticula wall. The diverticula wall is composed of mature collagenous connective tissue and lined in places by flattened epithelium. There is mild inflammatory reaction consisting of neutrophils and lymphocytes (arrow). Hematoxylin and eosin staining. ×20 magnification

Specific outcome in Pugs is reported in few studies.[3–6,15,16] Improvement after surgical management of SAD is reported in 66%-75% of cases, with follow-up periods ranging from 2 months to a few years.[3–6,15,16] Follow-up of longer than 6 months is reported only in 6 cases,[3,4,6,15,16] whereas follow-up of 12 months or longer is reported in 4 Pugs after surgical management of SAD.[6,15,16] Three of these Pugs had a recurrence of signs after initial improvement. Although the number of reported long-term outcome in Pugs is low, the results are in line with the findings of our study.

A moderate negative correlation (r = 0.50) was detected between the duration of clinical signs before presentation and the onset of recurrence of signs of neurological dysfunction, which differs from the results of the previous study.[12] On the other hand, shorter duration of signs showed a trend for prediction of better outcome after surgical procedure in a study with 7 dogs suffering from SAD.[16] Experimental studies showed that rats with longer lasting stationary chronic spinal cord compression have irreversible damage of the myelon because of exhaustion of the compensatory mechanisms, which might be true for dogs as well.[19] Current study findings might be used to recommend Pug owners to perform the surgery in a shorter period of time after SAD diagnosis. Intraoperative marsupialization and dissection of leptomeningeal adhesions did not influence the outcome in the current study. Marsupialization association with outcome was not statistically evaluated in previous studies, but the procedure was encouraged in a few previous publications.[3,16,20] On the other hand, other authors suggest that leptomeningeal adhesion dissection is a more important positive outcome predicting factor,[6] but currently there is no supporting data.

The etiology of poorer long-term outcome after surgical correction of SAD in Pugs compared to other breeds is not clear. Possible explanations include conformation of the vertebral column in this breed, and a high rate of concomitant IVDD might play a role in the higher recurrence rates. Only in one third (8/24) of dogs no concomitant pathology of the vertebral column was detected. On the other hand, complete evaluation of the vertebral column using computed

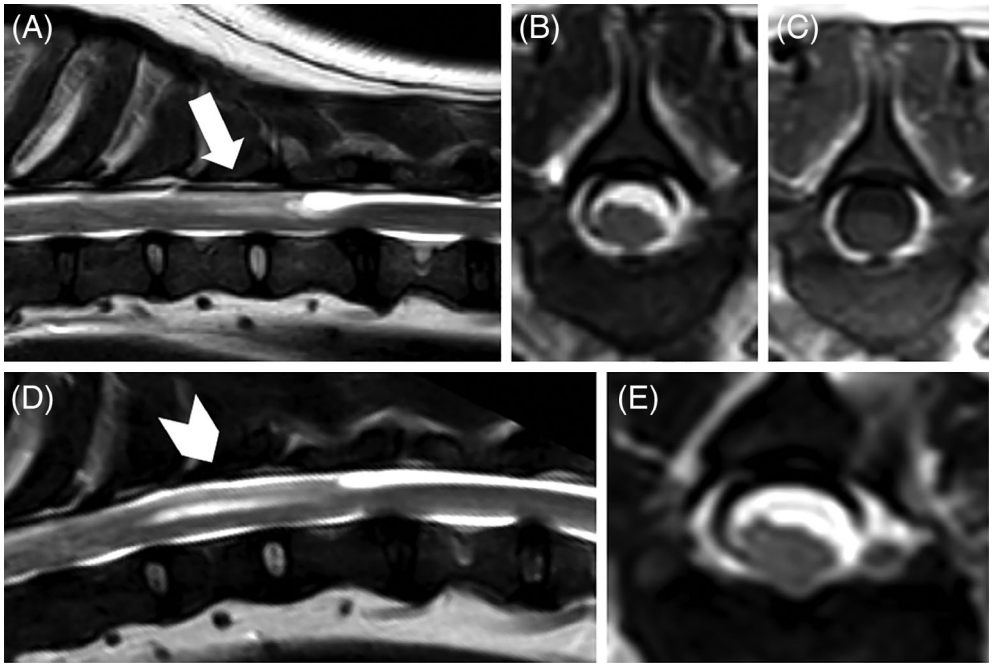

FIGURE 2 Pug, 7 years old, male. Initial mid-sagittal (A), transverse T2W (B), and T1W transverse (C) magnetic resonance images (MRI) demonstrating a spinal arachnoid diverticula (SAD) at T12-13 vertebral level. A follow-up MRI 7 months after surgery, demonstrating recurrence of the SAD in T2W mid-sagittal (D) and transverse (E) MRI images. Please note intramedullary hyperintensity which is already present in presurgical MR images (arrow) (A). Advanced intramedullary T2W hyperintensity in postsurgical MR images (arrowhead) (D)

tomography (CT) or at least radiographs were not performed in the mentioned 8 dogs, precluding exclusion of vertebral column anomalies, such as articular joint dysplasia, which is very common in the breed.[21] Disc degeneration is associated with the biomechanical changes of the vertebral column.[22–24] Pugs have higher rate of hemivertebra in their thoracic spine associated with signs of neurological dysfunction compared to other screw-tailed brachycephalic breeds.[25] In 2 case series, 3 of 5 Pugs had hemivertebrae and 1 of 5 Pugs had spondylosis deformans in the vertebra associated with SAD.[4,6] Facet joint malformations of the vertebral column in Pugs are associated with constrictive myelopathy.[26] Although in the study of Pugs without signs of neurological dysfunction, 97% of them had thoracic vertebral articular process aplasia or hypoplasia,[21] dogs in this investigation were younger in comparison to our study population raising the suspicion that clinical sequela of this malformation might occur later in life. Therefore, Pugs are predisposed for thoracic spine malformations which might lead to secondary myelopathies including formation of SAD.

In 58% of cases, IVDD was detected in the associated vertebra. Our findings differ from findings of other studies with various dog breeds suffering from SAD involved, but our findings are similar compared to studies where Pugs were investigated. Intervertebral disc disease is a rare concurrent finding in dogs of other breeds affected with SAD with prevalence ranging from none to 7%.[3,12,18] In contrast, IVDD was found in 60% (3/5) and 40% (2/5) of Pugs suffering from SAD in 2 studies.[4,6] Concomitant SAD and IVD herniation seem to have a higher prevalence in Pugs compared to other breeds possibly because of the previously discussed conformation of the vertebral column, which might result in increased mobility, accelerating disc degeneration, and protrusion.

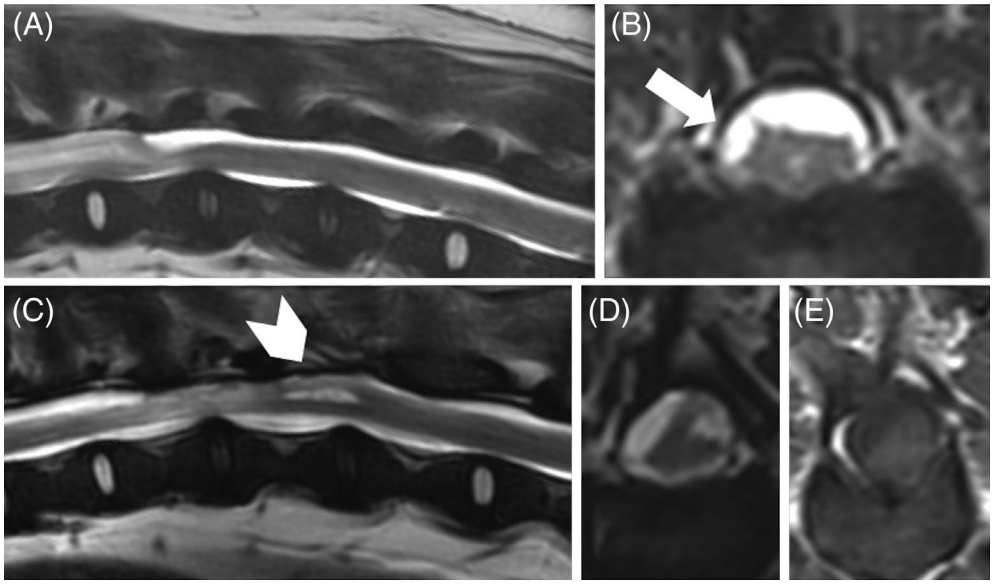

FIGURE 3 Pug, 6 years old, male. Initial mid-sagittal (A) and transverse T2W (B) magnetic resonance images demonstrating a multilobular spinal arachnoid diverticula (SAD) at T12 vertebral level. A follow-up magnetic resonance imaging (MRI) 20 months after surgery, demonstrating a new SAD formation originating from 1 part of the multilobular SAD (arrow), which was not addressed during the surgery, in mid-sagittal (C), transverse T2W (D), and T1W transverse (E) MR images. Advanced intramedullary T2W hyperintensity is present (arrowhead)

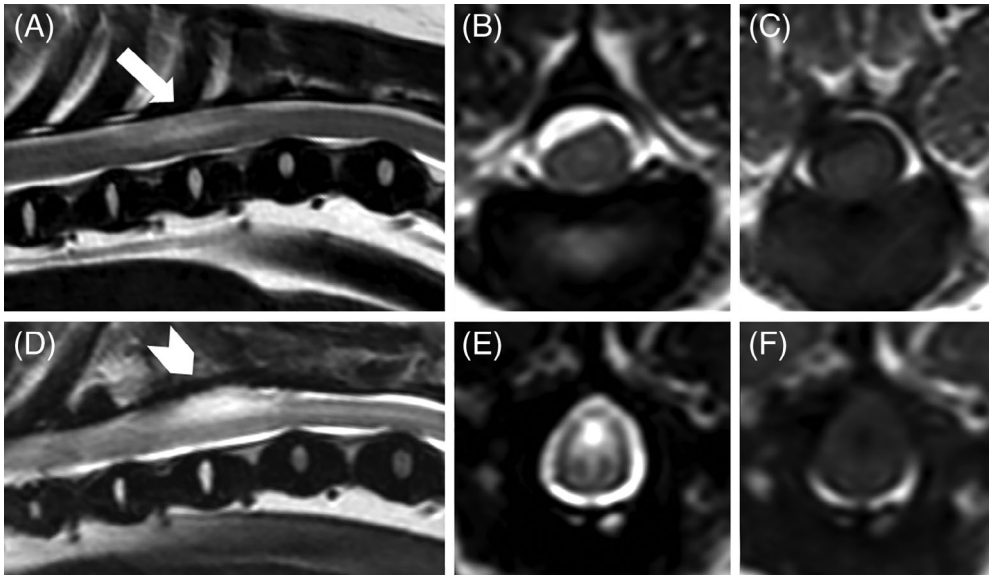

FIGURE 4 Pug, 5 years old, female. Initial mid-sagittal (A), transverse T2W (B), and T1W transverse (C) MRI images demonstrating a spinal arachnoid diverticula (SAD) at T9-10 vertebral level. A follow-up magnetic resonance imaging (MRI) 8 months after surgery demonstrating advanced T2W intramedullary hyperintensity (arrow) in T2W mid-sagittal (D), transverse (E), and T1W transverse (F) MRI images. Please note intramedullary hyperintensity which is already present in presurgical MR images (arrow) (A). Advanced intramedullary T2W hyperintensity in postsurgical MR images (arrowhead) (D)

Alternatively, the higher rate of IVDD might be related to a strong chondrodystrophic phenotype in Pugs.

One of the limitations of the current study was the inability to evaluate the presence of all possible vertebral column malformations because of the low-field MRI studies performed in the majority of cases. Another limiting factor was thick image slices. In cases where high-field MRI was performed, T1W images were available only in few cases precluding identification of subtle vertebral malformations. In addition, CT examination was unavailable in the study cases. Computed tomography is a preferred imaging modality to evaluate vertebral column abnormalities because it provides high contrast between bony structures and soft tissues as well as precise delineation of the facet joint surface.[27,28] In 6 cases, imaging data was sufficient to evaluate the vertebral malformations, and in all of them vertebral articular joint dysplasia associated with SAD was detected.

A second limitation is the lack of a detailed description of surgical procedures performed by various surgeons based on the surgeon's preference and documentation of surgical findings. It is not clear whether adhesions between pia mater and arachnoid were present in cases where it was not mentioned in the surgical reports. In addition, it is in question, if these adhesions were appropriately addressed in each case intraoperatively. In addition, the outcome after surgical treatment of SAD was evaluated at the same institutions where the surgery was performed only in limited amount of cases; otherwise, it was evaluated subjectively utilizing telephone questionnaire form, which is not as accurate as validated neurological score.

Histological examination findings of the excised dura mater did not differ from the other studies. Overall histological studies of intraoperatively resected dura mater in most cases reveal connective tissue proliferation, fibrosis, at some cases, infiltration with inflammatory cells, adhesions between pia mater and arachnoid or normal dura matter. These findings do not differ between cervical and thoracolumbar SADs.[3,11,13,14,18,29] Previously described histological examinations of Pug SADs in most cases revealed dura mater fibrosis and in 1 case—adhesions between pia mater and arachnoidea.[4,6,15] In addition, postmortem histopathological examination results of 1 study case did not differ from previous study findings involving various breed dogs as well, revealing dura mater fibrosis, infiltration with inflammatory cells, adhesions between pia mater and arachnoid, and secondary myelopathy consisting of Wallerian degeneration, axonal swelling, and syringohydromyelia of the adjacent white matter.[10,11,29] To our knowledge, postmortem histopathological changes are not described specifically for Pugs affected with SAD until now. Frequently detected dura mater fibrosis might be the result of continuous trauma to the dura mater which might result from abnormal, possibly increased, motion between adjacent vertebra. That would also explain the presence of infiltration of inflammatory cells and adhesion formation. In the cases of inflammatory cell infiltration in the resected dura mater, different treatment duration with nonsteroidal and steroidal anti-inflammatory drugs did not influence the outcome in the study Pugs presuming that dural inflammation is rather a consequence and contributing factor, but not the cause of the condition. In addition, there was no association found between perioperative prednisolone use in overall study population and the outcome. Another possible explanation of the changes detected in the leptomeninges might be that they occurred secondary to a gradual distention. It seems less likely that SAD in Pugs is only a congenital abnormality because of late age of onset; other factors should play at least a certain role in the development of SAD in Pugs.

Follow-up MRI was performed in 8 dogs. Recurrent or new SAD formation was present in 50% (4/8) of dogs in our investigation. This complication was detected in only 2 of 7 dogs in another study.[16] Surgery performed on recurrent SAD seem to resolve the clinical signs for unknown period of time.[16] A surgical technique was introduced to address the leptomeningeal adhesions by shunting the affected area.[30] Although some dogs treated with this surgical method still had persistent signs of neurological dysfunction and the follow-up period in the majority of cases was quite short, Pugs included in the study showed quite positive outcome.[30] Herniation of the spinal cord through the laminectomy defect and laminectomy membrane formation are observed in follow-up MRIs of dogs with recurrent signs of neurological

dysfunction.[12,16] In a couple of cases, these changes were managed surgically with improvement of the clinical signs.[12,16] We did not observe this kind of changes in our follow-up MRI. In the current study, increased intramedullary hyperintensity was observed in 6 of 8 cases, and in the majority of them, an intramedullary hyperintensity to a lesser extent was present already in preoperative MRI. Although intramedullary lesion is likely to progress in surgical cases where SAD was not appropriately excised, explanation of this observation is not clear in cases with seemingly complete removal of SAD. Progressive intramedullary hyperintensity in the absence of SAD might result from persistent leptomeningeal adhesions, hypermobility of the adjacent vertebras because of dysplastic facet joints, fibrotic tissue formation, or a combination of the above mentioned, in surgically managed cases. Alternatively, Pugs might have progressive signs of neurological dysfunction and myelopathy despite complete spinal decompression. Ongoing spinal cord degeneration because of neuronal necrosis, apoptosis, and gliosis might occur as a consequence of a chronic compression. Deactivation of compensatory mechanisms and nonreversible signs of neurological dysfunction in animals with chronic compressive lesions are described in the experimental studies, in 1 of them, chronic nonprogressive spinal cord compression causing ongoing deterioration of signs and progressive loss of motor neurons observed for a 25-week period after induction of compression.[19,31] Increase and formation of intramedullary lesions is known in chronic cases after acute spinal cord injury, it is in question if similar changes could develop after chronic compression.[32] To determine the relevance of increased T2W hyperintensity on postoperative MRI, a comparison with imaging results of Pugs without clinical deterioration would be necessary.

In summary, SAD has a favorable short-term but poor long-term prognosis after currently used surgical treatment. We suspect that various pathophysiological mechanism might contribute to this outcome including spinal deformity, vertebral column hypermobility, abnormal cerebrospinal fluid flow in the subarachnoid space, and unknown progressive intramedullary mechanisms. We propose that before planning a surgery, an MRI and CT examinations should be performed to investigate possible concurrent vertebral column abnormalities, which have the potential to contribute to SAD pathogenesis. Because these abnormalities could result in dynamic mechanical compression, additional stabilizing procedures should be considered in such cases. In addition, during the surgery, an attempt should be made to remove the dilated bulging dura mater as widely as possible (preferably reaching normally appearing pachymeninges), leptomeningeal adhesions should be carefully dissected, and marsupialization should be performed, but the efficacy of these procedures remains to be explored.

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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.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

There is IACUC approval from the Department of Small Animal Surgery, Neurology Service, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland; Referral Animal Hospital Aisti, Vantaa, Finland; and Dick White Referrals, Six Mile Bottom, UK.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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