veterinary sciences

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A Standardized Magnification-Assisted Microsurgical Approach for Canine Spinal Arachnoid Diverticulum: Retrospective Short-Term Outcomes in 11 Dogs

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Simple Summary

Some dogs develop a condition in which fluid accumulates around the spinal cord, causing pressure that can lead to progressive weakness and difficulty walking. This study aimed to describe the affected dog population, outline a standardized surgical treatment, and assess short-term recovery. Eleven dogs with gradually worsening movement problems were included after diagnosis was confirmed using advanced imaging. All dogs were treated with the same surgical procedure, which involved removing a small section of bone over the spinal cord and carefully opening and clearing the surrounding tissue to relieve pressure. All dogs but one showed improvement in their neurological function within one month after surgery. These findings indicate that this surgical approach is a feasible and effective method for short-term recovery in dogs with this condition. Although longer-term outcomes were not evaluated, the results provide useful information for veterinarians and contribute to improving treatment strategies and quality of life for affected animals.

Abstract

Spinal arachnoid diverticulum is a fluid-filled dilation within the subarachnoid space that can result in compressive myelopathy, prompting investigation into optimal surgical management and outcomes. This retrospective study reviewed clinical records of dogs treated surgically between January 2021 and July 2025, including 11 cases that met the criteria of chronic progressive para- or tetraparesis, MRI-confirmed diagnosis, and use of a standardized surgical technique. All dogs underwent dorsal laminectomy followed by durotomy, durectomy, and microsurgical dissection of adhesions performed by the same surgeon, with optical magnification and specialized instruments to ensure consistency. The condition was more common in males, with French Bulldogs overrepresented (n = 6); lesions were thoracolumbar in small breeds and cervical in the single large-breed dog. Short-term outcomes, assessed one month postoperatively, showed neurological improvement in all but one dog. These findings suggest that the described microsurgical approach is feasible and associated with favorable short-term neurological recovery in most cases. However, the absence of long-term follow-up limits definitive conclusions, and further studies are needed to evaluate sustained outcomes.

Keywords: spinal arachnoid diverticulum; dorsal laminectomy; durectomy; durotomy; dog

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Academic Editors: Ziyao Zhou and Zhijun Zhong

Received: 17 April 2026
Revised: 30 May 2026
Accepted: 1 June 2026
Published: 5 June 2026

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1. Introduction

Spinal arachnoid diverticulum (SAD) is a fluid-filled dilation in the subarachnoid space that can lead to compression of the spinal cord (SC) and associated myelopathy [1]. It is considered rare in dogs, with a breed predisposition in Pugs, French Bulldogs and Rottweilers [1]. Its etiopathogenesis is incompletely understood and is presumed to be multifactorial. Proposed factors include hereditary and congenital causes, biomechanical influences, prior or current spinal disorders such as intervertebral disc (IVD) protrusion or extrusion, and inflammatory diseases like meningomyelitis. One proposed mechanism involves the septum posticum, a dorsal leptomeningeal membrane connecting the pia mater and arachnoid. Altered cerebrospinal fluid (CSF) flow or injury-related thickening of these trabeculae may promote fibrous tissue formation and the development of SAD, particularly in the dorsal subarachnoid space, although this theory does not explain ventral or intracranial lesions [2–4]. Distinguishing congenital from acquired lesions remains challenging, and the variable age of affected dogs further complicates it. Diagnosis at a young age, the presence of vertebral malformations, or the absence of concurrent spinal pathology may suggest congenital origin, whereas preceding spinal injury, chronic IVD disease, or inflammatory conditions may suggest acquired forms [1,5–9].

Clinical signs typically reflect compressive myelopathy and include ataxia, paresis and pseudo-hypermetria, most often bilaterally due to the midline location of the diverticulum [1]. Neurological deterioration is usually slowly progressive [9,10], although asymptomatic cases have also been reported [11]. Urinary incontinence (UI) [9,10,12–14] and fecal incontinence (FI) [9,10,13,15–19] may occur in some patients. Diagnosis requires advanced diagnostic imaging techniques, such as magnetic resonance imaging (MRI) or computed tomography (CT) with myelography [1]. However, their accessibility and high cost may limit the recognition of true prevalence.

In dogs, SADs are most commonly described in the cranial cervical and caudal thoracic regions of the SC [20], especially at C3 and T12 [6–9,15,21]. This distribution corresponds to highly mobile spinal segments [16]. They are typically located dorsally within the median subarachnoid space [10,22,23], although ventral, lateral, or combined configurations have also been described [1]. They usually present as solitary lesions, although multiple diverticula have been reported [6,7,10,13,24]. Some SADs may remain undetected due to imaging of selected spinal segments, and when identified, they may represent incidental findings without clinical relevance [1]. Rottweilers, however, appear predisposed to multiple or bi-lobar SAD, particularly in the cervical region [6,8,20].

Both dorsal laminectomy and hemilaminectomy are used to expose SAD, depending on the position of the diverticulum and required surgical exposure [25]. Multiple surgical techniques have been described for SAD resolution, including durotomy [26], durectomy [9,13,16,19,26], dural marsupialization [9,19,26,27] and intra-arachnoid shunting [9,26]. Surgical interventions aim to decompress the SC by removing the diverticulum and adhesions, and to minimize recurrence [28,29]. Due to the low incidence of the disease, the heterogeneity in SAD location, and the diversity of surgical techniques applied, it remains difficult to draw firm conclusions regarding proper treatment.

Although surgical management of SAD has been previously described in the veterinary literature, detailed reports of a fully standardized microsurgical protocol performed by a single surgeon using dedicated ophthalmic microsurgical instruments and head-mounted loupes for optical magnification remain limited. The present study therefore aimed not only to report outcomes, but also to describe a reproducible surgical workflow designed to maximize precision during intradural dissection.

2. Materials and Methods

2.1. Patient Collection

This study retrospectively reviewed medical records of dogs diagnosed with SAD and surgically treated at the authors' institution between January 2021 and July 2025. Informed consent was obtained from all owners for data inclusion. Due to the retrospective nature of the study, formal Animal Ethics Committee approval was not undertaken. Inclusion criteria required complete medical records containing signalment, clinical history, findings of a general clinical examination, and an initial neurological examination including data on UI and FI. Only dogs with SC compression confirmed on MRI, specifically caused by a dilation of the subarachnoid space, were eligible for inclusion. All included cases had to be surgically treated using an identical surgical technique performed by the same surgeon. Records of postoperative neurological examinations were required to enable outcome assessment. Dogs with incomplete clinical and imaging records, dogs that underwent alternative surgical procedures, and dogs lacking postoperative evaluation records were excluded. Neurological status was graded using the modified Frankel scale as described by Mól et al. [30]: grade 0 (neurologically normal), grade 1 (spinal pain without neurological deficits), grade 2 (ambulatory paraparesis/ataxia), grade 3 (non-ambulatory paraparesis), grade 4 (paraplegia with intact nociception), or grade 5 (paraplegia with absent nociception). Spinal palpation was performed as a part of neurological examination to determine presence of pain. Follow-up neurological evaluations were performed daily during hospitalization and again one month postoperatively. Short-term outcome was considered positive if there was an improvement in gait, defecation and urination status or any combination of the above-mentioned depending on whether incontinence was present preoperatively or not. Lack of improvement or deterioration of neurological signs was considered a negative short-term outcome. Postoperative complications were categorized as minor if they were self-limiting or medically manageable, whereas life-threatening events requiring urgent surgical or medical intervention were classified as major complications [30].

2.2. Diagnostic Imaging

All dogs underwent MRI of the SC with intravenous contrast medium, targeted to the thoracolumbar or cervical region according to the neuroanatomical localization determined during the initial neurological examination. Because on-site MRI is not available at our institution, all dogs underwent imaging at external imaging centers. As a result, imaging protocols could not be standardized or influenced by the authors, and information about protocols used during imaging was limited to what was documented on external reports. However, all studies included at minimum T1-weighted and T2-weighted sequences of the affected spinal region, though the complete set of acquired sequences varied among patients. All MRI studies were interpreted by a board-certified diagnostic imaging specialist.

2.3. Anesthesia and Analgesia

Standard anesthetic protocol for surgery consisted of intramuscular (IM) premedication with methadone (Comfortan®, 10 mg/mL, Eurovet Animal Health B.V., Bladel, The Netherlands) at 0.2 mg/kg and dexmedetomidine (Sedadex® 0.5 mg/mL, Eurovet Animal Health B.V., Bladel, The Netherlands), whose dose was adjusted to the anesthetist's discretion (3–5 µg/kg IM). After the onset of sedation, an intravenous (IV) catheter was placed, and anesthesia was induced with ketamine (Ketamidor®, 100 mg/mL, VetViva Richter GmbH, Wels, Austria) at 0.5 mg/kg IV and propofol (Propofol-Lipuro 10 mg/mL, B. Braun Adria d.o.o., Zagreb, Croatia) to effect. All dogs were intubated with an endotracheal tube of appropriate size and connected to the anesthetic machine delivering isoflurane (Isofluran Piramal, Piramal, Critical Care B.V., Voorschoten, The Netherlands)

at 2% in 100% oxygen, titrated as needed to maintain adequate anesthetic depth. Fluid therapy consisted of isotonic balanced crystalloid infusion (Hartmann's solution® 500 mL, B.Braun Adria d.o.o., Zagreb, Croatia) at 5 mL/kg/h IV. Anesthesia was maintained with isoflurane delivered in 50–60% oxygen and a continuous rate infusion (CRI) of ketamine (5–10 µg/kg/min) with or without dexmedetomidine (0.5–2 µg/kg/h). Intraoperative rescue analgesia consisted of ketamine boluses (0.3 mg/kg IV) and/or fentanyl (2 µg/kg IV). The anesthetic protocol was adjusted based on age and comorbidities as needed. Physiological parameters were monitored continuously using a multiparameter monitor (Mindray ePM 12M Vet, Szenzhen Mindray Animal Medical Technology, Ltd., Shenzhen, China) with values recorded every 5 min.

2.4. Surgical Technique

All surgeries were performed by the same surgeon to minimize technical variability. The surgical approach was selected based on surgeon experience and institutional preference. Dogs were positioned in sternal recumbency, and a standard surgical field preparation protocol was applied. All dogs underwent dorsal laminectomy accompanied by durotomy, durectomy and excision of SAD. Optical magnification was used throughout the procedure via head-mounted surgical loupes with integrated lighting and 3× magnification (Tailor-made loupes, Univet Loupes Spa, Rezzato, Italy). Dorsal laminectomies were performed using a high-speed neurosurgical drill (Aesculap® ELAN 4 electro, Aesculap AG, Tuttlingen, Germany), and the surgical window was further enlarged using Kerrison rongeurs. Upon exposure of the SC and visualization of SAD, a durotomy was performed using an ophthalmic microsurgical knife (Stab knife 15°, Sidapharm P.C., Thessaloniki, Greece). Durectomy was performed using angled Castroviejo corneal scissors to expose the SAD, followed by blunt dissection of SAD adhesions using a nerve hook (Figure 1a). Following exposure, SAD was incised using a curved-tip injection needle (23 G) (Figure 1b) and excised using Vannas straight and curved micro scissors. The dura was not sutured, and no synthetic or biological dural substitute, autologous fat graft, or hemostatic material was used following durectomy. No spinal stabilization technique was applied. The surgical field was routinely closed in layers.

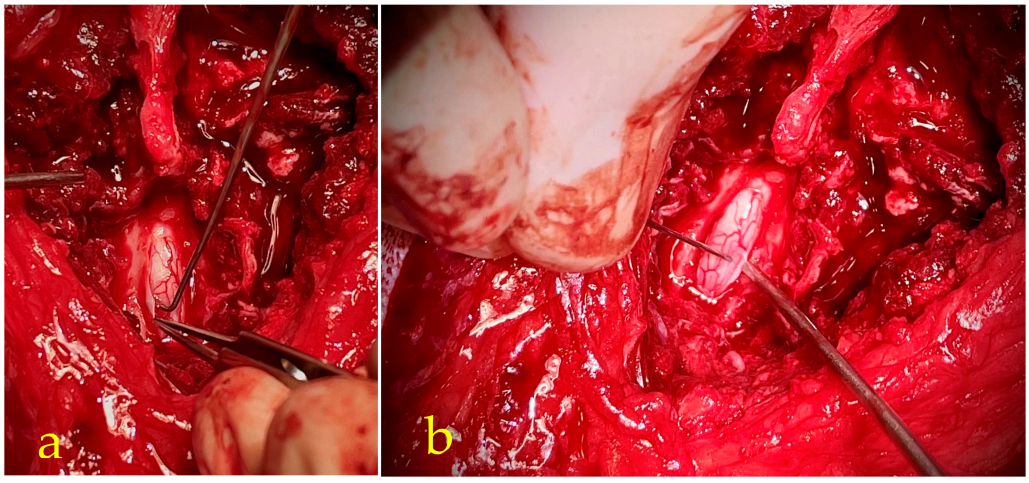


Figure 1. Microsurgical management of SAD with (**a**) blunt dissection of SAD adhesions using a nerve hook; (**b**) incision of the SAD using a curved tip injection needle.

2.5. Postoperative Care

Dogs were hospitalized over several days for postoperative monitoring, analgesia, anti-inflammatory therapy, and bladder management. During hospitalization, they received isotonic balanced crystalloid fluid therapy (Hartmann's solution® 500 mL, B.Braun

Adria d.o.o., Zagreb, Croatia) at maintenance rate of 60 mL/kg/day. Pain management consisted of methadone at 0.15 mg/kg IV every 4–5 h and gabapentin (Katena®, Belupo Pharmaceuticals and Cosmetics d.d., Koprivnica, Croatia or Gabapentin oral solution 5%, Syrspend®, 50 mg/mL, Ljekarna Štefanić Pharmaceutical Laboratory, Đurđevac, Croatia) at 10 mg/kg orally every 8 h. Anti-inflammatory therapy consisted of either a non-steroidal anti-inflammatory drug (NSAID) or a corticosteroid. The two drug classes were never combined or alternated, and the choice of agent often depended on the medication the animal had already been receiving prior to referral to our institution. Therapy with NSAIDs included either meloxicam (Meloxidolor®, 5 mg/mL, Le Vet. Beheer B.V., Oudewater, The Netherlands) administered at 0.2 mg/kg IV on the first day, followed by 0.1 mg/kg IV once daily, or carprofen (Rycarfa®, 50 mg/mL, Krka Farma d.o.o., Jastrebarsko, Croatia) at 4.4 mg/kg IV once daily. When corticosteroids were used, dexamethasone (Dexamethasone®, 2 mg/mL, Alfasan International B.V., Woerden, The Netherlands) was administered at 0.1–0.2 mg/kg IV once daily. Antimicrobial prophylaxis consisted of cefazolin (Zepilen®, 1 g, Medochemie Ltd., Limassol, Cyprus) at 22 mg/kg IV every 8 h over the first 24 h postoperatively. A fentanyl patch (Durogesic®, Johnson & Johnson S.E. d.o.o., Zagreb, Croatia) was applied postoperatively at 3–5 µg/kg/h and left for 4 days. Methadone was discontinued once the fentanyl patch started to take effect. When required, urinary catheterization was performed to maintain hygiene and ensure complete bladder emptying. Patients were discharged once pain was controlled and they regained voluntary urination. Owners received written instructions regarding activity restriction, neurologic monitoring, and medication administration. Follow-up neurological examinations were scheduled for one month after surgery.

3. Results

A total of eleven (n = 11) dogs were found in a search of archived data, and all of them met the inclusion criteria. Males comprised 8/11 (73%) of included dogs, and females 3/11 (27%). All females were spayed, while one male was neutered. Patient age ranged from 2 to 9 years. The mean age of female dogs was 8.0 ± 1.0 years (range: 7–9 years), while the mean age of males was 5.63 ± 2.88 years (range: 2–9 years). The overall mean age, regardless of sex, was 6.27 ± 2.69 years. Five different breeds were represented: French Bulldog (n = 6; 55%), Pug (n = 2; 18%) and one each of German Boxer, West Highland White Terrier (WHWT) and Yorkshire Terrier (each n = 1; 9%).

3.1. Clinical Data

Chronic slowly progressive neurological signs were reported in all dogs, with durations ranging from 2 to 24 months. According to owner reports, the two youngest dogs (German Boxer and WHWT), both aged 2 years, developed initial clinical signs at approximately one year of age, while other dogs showed symptom onset later in life (range 4–9 years). In cases with thoracolumbar SAD, paresis affected the pelvic limbs (paraparesis); in the cervical form, all four limbs were involved (tetraparesis). According to the modified Frankel scale, upon first neurological examination, most dogs (10/11, 91%) were scored as grade 2, with only one dog as grade 4 (Pug). Discomfort during palpation of the thoracolumbar region was documented in 1/11 dogs (9%; WHWT), and in 1/11 dogs (9%; French Bulldog) the owner reported noticing signs of discomfort or pain when early clinical signs appeared. However, during neurological examination on admission, the dog did not exhibit any signs of pain. Urinary incontinence was observed in 4/11 (36%) dogs, two of which (2/11, 18%) also exhibited signs of FI.

3.2. Imaging Findings

A characteristic teardrop-shaped lesion was described in 8/11 dogs (73%), as shown in Figure 2a, although a diagnosis of SAD was confirmed in all cases (100%). In 1/11 dogs (9%), the lesion was described as having a "tepee-like" appearance, while in 2/11 dogs (18%) it was described as a subarachnoid space dilation typical for SAD. In all cases, SAD was located dorsally. The position was described as midline in 2/11 dogs (18%) (Figure 2d), dorsal with mild left lateralization in 4/11 dogs (36%) (Figure 2c), dorsal with marked left lateralization in 2/11 dogs (18%) (Figure 2b), dorsal bilateral in 2/11 dogs (18%) (Figure 2e) and dorsal with marked right lateralization in 1/11 dogs (9%). With respect to vertebral segment location, SAD was identified at the following sites: T13 (1/11; 9%), T11 (2/11; 18%), T10–T11 (1/11; 9%), T10 (1/11; 9%), T9–T10 (2/11; 18%), T9 (1/11; 9%), T8–T9 (1/11; 9%), T7–T8 (1/11; 9%), and C4 (1/11; 9%). Accordingly, 10/11 of dogs (91%) exhibited thoracic SC involvement and they were all small breeds, while the only large-breed dog (Boxer) had a cervical lesion. Additional MRI findings included suspected SC edema in 8/11 dogs (73%) and/or syringohydromyelia (SHM) in 8/11 dogs (73%). Intervertebral disc protrusion with concurrent disc degeneration was observed in 7/11 dogs (64%), while only IVD degeneration without any protrusions was observed in 1/11 dogs (9%). All French Bulldogs in our cohort showed IVD pathology (IVD degeneration and/or protrusion), and vertebral malformations were identified in 5/6 (83%) of them. Notably, French Bulldogs were the only breed in which vertebral malformations were detected on MRI. These findings reflect only the spinal segments that were imaged based on clinical neuro-localization. All of the MRI findings for each dog are summarized in Table 1.

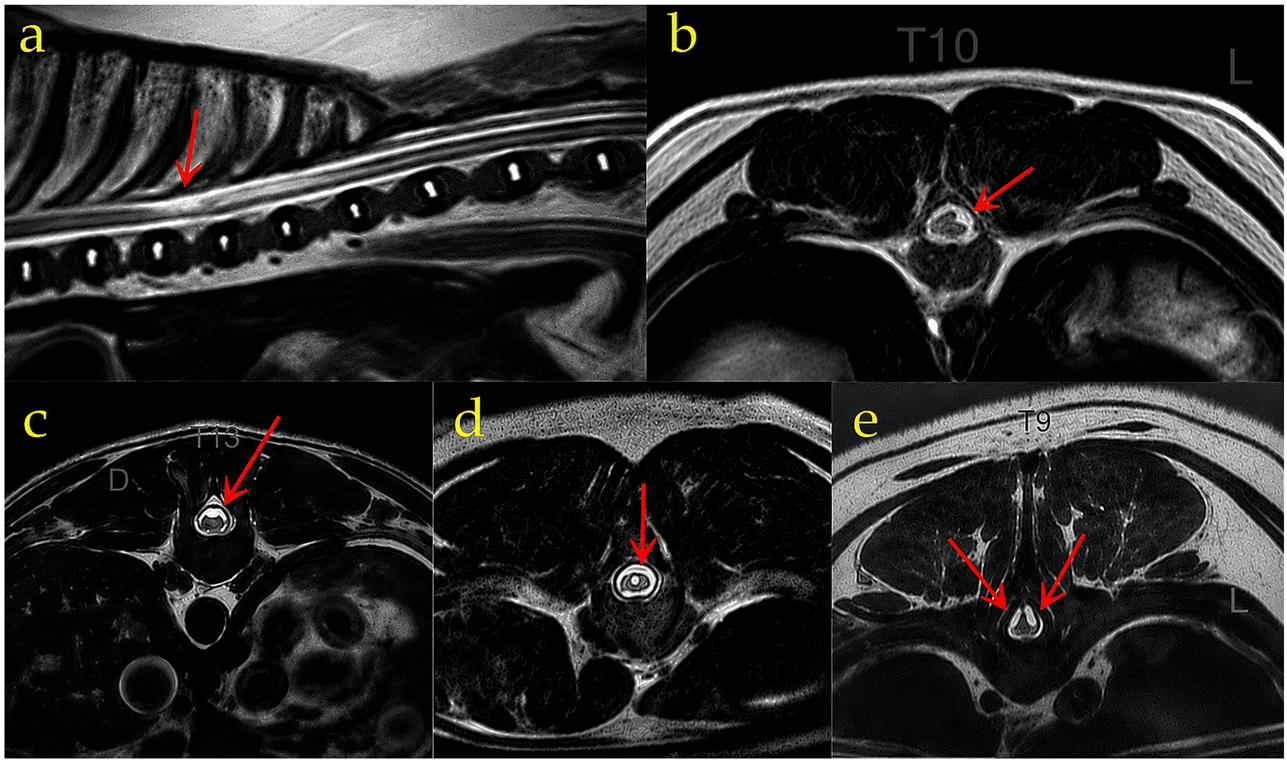


Figure 2. MRI appearance and positional variations in SAD in dogs: (**a**) sagittal MRI scan of thoracic spine showing a teardrop-shaped enlargement of the subarachnoid space at the level of T7–T8; (**b**) transverse MRI scan of thoracic spine showing dorsal position of SAD with marked lateralization at the level of T10; (**c**) transverse MRI scan of thoracic spine showing dorsal position of SAD with mild lateralization at the level of T13; (**d**) transverse MRI scan of thoracic spine showing a midline position of SAD at the level of T11; (**e**) transverse MRI scan of thoracic spine showing bilateral dorsal position of SAD at the level of T9. Red arrows indicate SAD.

Table 1. Summary of all MRI findings in dogs with SAD.

BREED	SAD LOCALIZATION	IVD CHANGES	VERTEBRAL MALFORMATIONS	SPINAL CORD LESIONS	SAD POSITION
German Boxer	C4	-	-	Cervicothoracic intramedullary lesion compatible with inflammatory condition or SHM	Dorsal
WHWT	T9–T10	-	-	Focal myelopathy (T10–T11) compatible with SC edema or early/developing SHM	Dorsal/ dorsolateral left
Yorkshire Terrier	T10–T11	Multiple IVD degenerations and mildly compressive protrusions in thoraco-lumbar region	-	-	Dorsolateral left
Pug	T7–T8	-	-	Focal myelopathy (T9–T10) compatible with SC edema or early/developing SHM	Dorsal/ dorsolateral left
French Bulldog	T11	Multiple IVD protrusions (T11–L3); Degeneration of all visible IVDs (T3–L7)	-	Intra-medullar hyperintensity (T11) compatible with edema/gliosis and mild SHM	Dorsal
French Bulldog	T11	Multiple minimally compressive IVD protrusions (T11–L7); Multiple IVD degenerations	Bilateral costovertebral fusion at T13; Asymmetric lumbosacral transitional vertebra with hypertrophic left transverse process of L7	Focal myelopathy (T8–T11) compatible with SC edema or early/developing SHM	Dorsal/ dorsolateral left
French Bulldog	T9–T10	Mild to moderate IVD protrusion (L3–L4); Degeneration of all visible IVDs	Hemivertebra (T8); Kyphosis	Focal myelopathy (T9) compatible with SC edema or early/developing SHM	Dorsolateral right
French Bulldog	T9	Multifocal IVD degeneration L7–S1 IVD protrusion with mild SC compression	Multiple hemivertebra; Kyphosis	-	Dorsolateral left
French Bulldog	T13	Multifocal IVD degeneration; Minimal non-compressive IVD protrusions (T12–L2)	Multiple thoracic and sacral vertebral congenital malformations leading to angular deviations of the corresponding spinal segments	Intradural extramedullary SC compression and segmental myelopathy (T9–T12) compatible with SC edema or early/developing SHM	Dorsal/ dorsolateral left

Table 1. *Cont.*

BREED	SAD LOCALIZATION	IVD CHANGES	VERTEBRAL MALFORMATIONS	SPINAL CORD LESIONS	SAD POSITION
Pug	T8–T9	Multifocal thoracic and lumbar IVD degenerations; Multiple thoracic and lumbar IVD protrusions	-	Segmental myelopathy (T10), possible SC edema or early/developing SHM	Dorsal bilateral
French Bulldog	T10	Multiple IVD degenerations	Multiple thoracic hemivertebra	Focal myelopathy compatible with SC edema/gliosis	Dorsal bilateral

C—cervical vertebra, IVD—intervertebral disc, L—lumbar vertebra, T—thoracic vertebra, SAD—spinal arachnoid diverticulum, SC—spinal cord, SHM—syringohydromyelia, WHWT—West Highland White Terrier.

3.3. Surgical Observations

In all dogs, intraoperative findings confirmed the same localization of SAD as described on preoperative MRI. Dorsal laminectomy was sufficient to provide adequate visualization of SAD in 10/11 dogs (91%). In one dog presenting with marked left lateralization of SAD (Yorkshire Terrier), the surgical field was extended to the left side to remove additional lamina covering markedly lateralized SAD and hence improve visualization and facilitate excision of SAD. No postoperative complications were encountered in any of the dogs.

3.4. Short-Term Outcomes

Duration of hospitalization ranged from 2 to 10 days (median 3 days). One month postoperatively, a positive short-term outcome was observed in 10/11 dogs (91%) with improvement in neurological status compared to preoperative findings. Although the modified Frankel scale was used for initial neurological grading, short-term postoperative neurological improvement was primarily described qualitatively. Since most dogs were ambulatory at presentation (modified Frankel grade 2), early postoperative clinical improvement did not necessarily correspond to a reduction in Frankel grade within the one-month follow-up period. Consequently, qualitative descriptors such as gait improvement were considered more representative of subtle neurological recovery during short-term follow-up. FI resolved in 1/2 dogs (50%), whereas UI resolved in 1/4 dogs (25%) and improved in 2/4 dogs (50%), according to the owners' reports. One dog (French Bulldog, intact male, SAD at T13 with non-compressive IVD protrusions at T12–L2, FI, UI) had a negative short-term outcome, as no neurological improvement was evident even one month after surgery. The summary of initial neurological status, duration of hospitalization and evaluation at one month postoperatively is presented in Table 2.

Table 2. Summary of patient data and short-term outcomes.

BREED	SEX	AGE (Years)	MODIFIED FRANKEL SCORE ON ADMISSION	FECAL AND URINARY INCONTINENCE	DURATION OF HOSPITALIZATION (Days)	FOLLOW-UP AT ONE MONTH POSTOPERATIVELY
German Boxer	M	2	2	-	4	Gait improved
WHWT	M	2	2	-	4	Gait improved
Yorkshire Terrier	M	4	2	-	2	Gait improved

Table 2. *Cont.*

BREED	SEX	AGE (Years)	MODIFIED FRANKEL SCORE ON ADMISSION	FECAL AND URINARY INCONTI-NENCE	DURATION OF HOSPITALIZA-TION (Days)	FOLLOW-UP AT ONE MONTH POSTOPERA-TIVELY
Pug	M	5	2	-	10	Gait improved
French Bulldog	FS	8	2	-	3	Gait improved
French Bulldog	M	9	2	-	3	Gait improved
French Bulldog	FS	9	2	-	6	Gait improved
French Bulldog	FS	7	2	UI	2	Gait improved UC
French Bulldog	M	6	2	FI, UI	3	No improvement
Pug	M	8	4	UI	4	Gait improved UI less severe
French Bulldog	MN	9	2	FI, UI	3	Gait improved FC UI less severe

FS—female spayed, FC—fecal continence, FI—fecal incontinence, M—male, MN—male neutered, UC—urinary continence, UI—urinary incontinence, WHWT—West Highland White Terrier.

4. Discussion

A variety of surgical techniques have been described for the management of SAD, each offering distinct advantages and limitations. Our approach most closely resembles those reported by Spinillo et al. [31] and Mól et al. [30], while incorporating several differences. All three techniques employ dorsal laminectomy, followed by durotomy and meticulous microsurgical adhesion removal under magnification. The benefit of durotomy is well established, as it facilitates SC decompression by releasing accumulated CSF [7,11,26], evidenced by significant CSF effusion following durotomy [9]. Whereas Spinillo et al. [31] and Mól et al. [30] used an operating microscope, and Bismuth et al. [16] reported the use of a surgical microscope or magnifying glasses, our procedures were performed using head-mounted surgical loupes with an integrated light source and 3× magnification. The magnification employed is reported here to provide a more detailed description of the surgical technique and facilitate reproducibility. Although the use of Castroviejo micro-surgical scissors has been described for durotomy [31], we employed them for durectomy. Even though the literature describes the use of iris scissors for durectomy [32], we opted for angled Castroviejo scissors to enhance precision and avoid damage to the underlying spinal cord. In our experience, the spring-loaded handles of Castroviejo scissors make them more ergonomic, providing superior tactile feedback and controlled movement for delicate, repetitive actions. Therefore, our technique integrates ophthalmic microsurgi-cal principles into intradural spinal surgery. Durectomy, involving excision of the dura surrounding the SAD, is considered by several authors to confer more sustained clinical im-provement [6,8,9,11,16,19,26]. A key difference between our method and the two previously described techniques lies in the management of the dural defect. We did not suture the dura following durotomy, as performed by Spinillo et al. [31], nor did we apply an artificial dural substitute following durectomy, as described by Mól et al. [30]. We intentionally omitted dural closure and marsupialization to avoid additional SC traction and the risk

of ventral subdural space occlusion [1]. The intentional omission of dural closure and marsupialization has previously been described by several authors [13,19,33,34]. Although marsupialization can maintain continuous CSF drainage by suturing the dura to adjacent paravertebral soft tissues [5,8,9,19,26–28], evidence for its clinical superiority remains inconsistent [13,33], and the procedure is technically demanding, with outcomes strongly dependent on surgeon expertise [1]. Furthermore, variability in the duration of reported long-term outcomes, as well as the use of different dural closure techniques, makes it difficult to draw conclusions about the superiority of any single technique [1]. Therefore, the impact of our decision to omit dural closure could only be adequately assessed through long-term follow-up, which was not feasible in this retrospective study.

Generally, short-term outcomes are favorable, with rapid recovery of walking and bladder/bowel function, which aligns with our findings, whereas long-term outcomes may be less favorable [6,7,9]. The only dog without short-term neurological improvement in our cohort had concurrent mild non-compressive IVD protrusions between T12 and L2 identified preoperatively. Although no intraoperative vertebral instability was observed, and spinal stabilization was not considered necessary in any of the dogs, it is possible that postoperative biomechanical alterations following dorsal laminectomy may have influenced adjacent disc positioning or spinal cord compression dynamics. However, because postoperative MRI was not performed, this hypothesis could not be confirmed and therefore remains speculative. Despite surgical management being the recommended treatment, yielding more noticeable improvement than conservative treatment [6,13], recurrence rates of up to 20% have been reported [9,22]. Recurrence may result from SAD relapse, membrane formation at the laminectomy site, or SC herniation through the dural defect at the surgical site [1,19], all of which require long-term follow-up for accurate assessment.

Previous studies on SAD in dogs have generally included relatively small cohorts, although several larger datasets have recently been published [22,26]. The largest multicenter study included 122 dogs over a 12-year period [10]. In our institution, surgically treated cases ranged from 1 to 4 dogs annually, reflecting the rarity of SAD in clinical practice.

A marked male predominance has consistently been reported, with approximately 80% of affected dogs being male [8,10,33], which aligns with our cohort (73%). Because the hormonal component has been proposed to influence CSF volume [35,36], its contribution to SAD pathogenesis remains plausible and merits further investigation, particularly as only one male in our study was neutered.

All dogs in our cohort were purebred and belonged to previously reported predisposed breeds [1,10], supporting earlier suggestions of hereditary and/or breed-associated influences in SAD development [5,37]. French Bulldogs were particularly notable, as all had concurrent MRI-detected spinal abnormalities and were the only breed exhibiting vertebral malformations, consistent with previous reports linking concurrent spinal pathology and SAD formation in this breed [1,33,38]. Our age distribution generally corresponded with previous studies, with thoracolumbar SAD predominating in older dogs and cervical lesions occurring at a younger age [9,10].

Most dogs with SAD exhibit slowly progressive neurological signs [9,10], which was also observed in our cohort. Although ataxia and pseudo-hypermetria are commonly described [7–10], paresis with proprioceptive deficits predominated in our dogs, likely reflecting later-stage presentation and the presence of concurrent spinal abnormalities in all cases. Fecal and/or urinary incontinence has been reported in approximately 11.5% of SAD cases [10], particularly in thoracolumbar lesions [13]. In our cohort, incontinence occurred in 4/11 dogs (36%), although mild cases may have gone unrecognized by owners. This likely reflects the disruption of ascending sensory pathways leading to impaired perception of rectal distension and defecation, as well as upper motor neuron urinary

sphincter dysfunction characterized by uninterrupted urine flow and post-void dribbling, consistent with urethral dyssynergia [1,9,10,15,23]. However, meaningful interpretation of factors contributing to the higher incidence of incontinence in our cohort is difficult given the small sample size.

The presence of spinal pain in dogs with SAD varies across studies [10,23,39]. Pain detected during examination or reported by owners occurs in approximately 10–19% of cases [1,6,9,10,14,17,40,41], consistent with our findings. In contrast, neuropathic pain associated with intradural spinal arachnoid cysts in humans is reported more frequently [42], highlighting the challenges of pain recognition in dogs.

SAD may also be underdiagnosed in dogs presenting with neurological signs. CT myelography and MRI remain the only reliable diagnostic modalities [1,9,10,13,23], but when CT identifies another abnormality such as IVD protrusion, further imaging capable of detecting SAD may not be pursued. This issue is particularly relevant in institutions without in-house MRI access, such as ours, where external referral may delay diagnosis. Consequently, some dogs with clinical signs compatible with SAD likely remain undiagnosed, particularly given the high prevalence of concurrent spinal abnormalities that may obscure the underlying condition. Indeed, 7/11 dogs (64%) in our study had concurrent IVD degeneration and/or mild multifocal protrusions. French Bulldogs commonly exhibited additional abnormalities, including SHM and vertebral malformations, consistent with previous studies [33,38]. Similarly high rates of SHM have been reported in dogs with SAD [34,43], and SC edema or early SHM was identified in 73% of our cohort. Although the causal relationship between SHM, SAD, and concurrent spinal disease remains unclear [1,43], collectively, these observations support the hypothesis that concurrent vertebral and SC abnormalities may contribute to SAD development and highlight breed-related predispositions to multifactorial spinal pathology [1].

The cranial cervical region is a commonly reported location for SAD [20], while thoracolumbar lesions are most frequently described between T9 and L1 [9,10,23]. Our findings were consistent with published distributions, with most lesions occurring in the thoracolumbar region, particularly in French Bulldogs. As previously proposed, SADs tend to develop in regions of increased spinal mobility [7–9]. All small-breed dogs in our cohort had thoracolumbar lesions, whereas the only large-breed dog had a cervical lesion.

The dorsal location of SAD, with or without lateralization, is most frequently described in the literature [6,8–10,16], and our findings were consistent with this pattern. Interestingly, we observed a predominance of left-sided lateralization, although its significance remains unclear given the small sample size. Precise MRI assessment of lesion location and lateralization is essential for surgical planning, as markedly lateralized lesions may require an extension of the surgical approach [44]. In one Yorkshire Terrier with pronounced left-sided lateralization, ipsilateral extension of the approach was necessary to improve visualization and facilitate safer instrument insertion and manipulation.

This study has several limitations, the most significant being the lack of structured long-term follow-up. Because postoperative MRI was not performed later in recovery for ethical and financial reasons, this limits the objective assessment of long-term changes and precludes reliable monitoring of recurrence. Furthermore, the sample size was small, and only one large-breed dog was included, restricting the generalizability of the findings. Additionally, all MRIs were performed at external institutions, meaning imaging protocols and sequences were not standardized or under our control, thereby potentially reducing comparability between patients.

5. Conclusions

This study demonstrated rapid postoperative recovery, with a median hospitalization of 3 days, and favorable short-term outcomes in 91% of dogs, without immediate postoperative complications. The use of ophthalmic microsurgical instruments and optical magnification facilitated the precise dissection of SAD, and spinal stabilization was not required. While no conclusions regarding optimal surgical techniques can be drawn, these findings contribute additional descriptive data on the clinical course and early postoperative outcome of dogs with SAD treated surgically. However, larger prospective studies with standardized imaging protocols and controlled long-term follow-up are required for better assessment of long-term neurological outcomes, recurrence rates and prognostic factors in dogs undergoing this type of surgical treatment for SAD.

Author Contributions: Conceptualization: P.D.; Methodology: P.D., B.P.; Software: Not applicable; Validation: Not applicable; Formal Analysis: P.R.; Investigation: P.R.; Resources: Not applicable; Data Curation: P.R.; Writing—Original Draft Preparation: P.R., P.D.; Writing—Review and Editing: P.R., P.D., B.P.; Visualization: P.R., P.D.; Supervision: P.D., B.P.; Project Administration: Not applicable; Funding Acquisition: Not applicable. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions as raw data include personal information about the owners and the dogs included.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CSF	Cerebrospinal fluid
FI	Fecal incontinence
IVD	Intervertebral disc disease
MRI	Magnetic resonance imaging
SAD	Spinal arachnoid diverticulum
SC	Spinal cord
SHM	Syringohydromyelia
UI	Urinary incontinence
WHWT	West Highland White Terrier

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