**LATE ONSET RECURRENCE OF NEUROLOGICAL DEFICITS AFTER SURGERY FOR SPINAL ARACHNOID DIVERTICULA**

E. Alcoverro DVM MRCVS1, J. F. McConnell BVM&S DVR DipECVDI CertSAM MRCVS1, D. Sánchez-Masian DVM DipECVN MRCVS1, L. De Risio DVM PhD DipECVN MRCVS2, S. De Decker DVM PhD MVetMed DipECVN FHEA PGCert Veted MRCVS3, R. Gonçalves DVM MVM DipECVN FHEA MRCVS1

1Institute of Veterinary Science, Small Animal Teaching Hospital, University of Liverpool, Leahurst, Chester High Road, Neston CH64 7TE, UK

2The Animal Health Trust, Lanwades Park, Kentford, Suffolk, CB8 7UU, UK

3Department of Clinical Science and Services, Royal Veterinary College, University of London, Hatfield Hertfordshire AL9 7TA, UK

**Email for correspondence: r.goncalves@liv.ac.uk**

**Abstract**

Spinal cord dysfunction secondary to spinal arachnoid diverticula (SAD) has been widely reported in the veterinary literature and there is some suggestion that surgical treatment may provide better outcomes than medical treatment. Despite this, previous reports have mentioned cases with recurrence of clinical signs following surgical treatment but the cause for this has not been further investigated.

The medical records of 7 dogs and 1 cat which presented for investigation of recurrence of neurological deficits at least 6 months after surgery for SAD were retrospectively reviewed. Median time to relapse of the neurological deficits was 20.5 months after surgery. On repeated imaging, 3/8 cases showed clear regrowth of diverticulum, 2/8 cases showed dorsal compression at the previous laminectomy site (presumed to be the laminectomy membrane), and 3/8 cases showed herniation of the spinal cord through the laminectomy defect associated with a stellate appearance to the spinal cord with small multiloculated areas of dilation of the subarachnoid space. Repeat surgical intervention was most successful in the cases where SAD recurrence was identified whilst medical treatment resulted in either subtle improvement or stabilisation on the clinical signs, sometimes followed by slow deterioration.

**Introduction**

Spinal arachnoid diverticula (SAD) have been given a plethora of names in both the human and veterinary literature such as meningeal cysts, arachnoid or subarachnoid cysts, leptomeningeal cysts and pseudocysts. All them refer to a single condition characterised by an accumulation of CSF and dilation of a focal area of the meninges, with most being congenital (primary), although some may be acquired (secondary) (Lowrie and others 2014). The word “cyst” is defined as a closed epithelium-lined sac or capsule containing liquid, air or semi-solid substance. Because these lesions lack an epithelial lining, the term spinal arachnoid diverticula (SAD) will be used in this manuscript, instead of cyst, which is considered a misnomer (Lowrie and others 2014).

Clinical signs of SAD consist in a chronic, progressive myelopathy with neurological signs that reflect the localisation of the lesion (Skeen and others 2003, Sugiyama and others 2009, Rohdin and others 2013, Mauler and others 2014, Adams and others 2015). Spinal hyperaesthesia is rarely seen in these patients (Mauler and others 2014, Adams and others 2015). SAD can develop at any spinal cord level, but they are commonly found in the cranial cervical region in large breed dogs whereas in small breed dogs, the thoracic segments are more commonly affected. In cats, SAD have almost exclusively been reported in the thoracolumbar region (Galloway and others 1999, Schmidt and others 2007, Surgiyama and Simpson 2009) with only one recentlyreported in the cervical region (Adams and others 2015).

Although not common in dogs, SAD is even rarer in cats. All cats with SAD reported in the literature had evidence of previous or concurrent spinal disease at the time of diagnosis of SAD (Grevel and others 1989, Shamir and others 1997, Galloway and others 1999, Vignoli and others 1999, Schmidt and others 2007, Surgiyama and Simpson 2009, Adams and others 2015), suggesting that acquired SAD may be more common than in dogs.

Magnetic resonance imaging (MRI) is the imaging modality of choice for diagnosis of SAD in people, although computed tomography (CT) myelography can establish whether there is communication of the cyst with the subarachnoid space (Nabors and others 1988, Flegel and others 2013). MRI is now widely used for the diagnosis of SAD in veterinary medicine with one study suggesting that the addition of a single‐shot turbo spin‐echo sequence gives a more than twofold increase in SAD detection compared to a T2‐weighted sequence alone (Seiler and others 2012).

Both medical and surgical treatments have been used for SAD in the veterinary literature.Medical management (i.e. glucocorticoid therapy) may in some cases result in stabilisation or even improvement of the neurological signs (Mauler and others 2017). Surgery though is often the treatment of choice.Surgical techniques described include marsupialisation (McKee and Renwick 1994) or fenestration of the dura (Frykman 1999, Rylander and others 2002), ideally associated with removal of visible subarachnoid adhesions. No predictors of good outcome have been identified, although there is a trend towards a better outcome in dogs less than 3 years of age, those that had duration of clinical signs of less than 4 months, and when marsupialization was used as the surgical technique (Skeen and others 2003). Another study suggested that durectomy might provide a more permanent improvement than durotomy and drainage alone (Frykman 1999).

A recent study described and compared medical and surgical treatments in dogs with SAD (Mauler and others 2017). Outcome was subjectively assessed by the owner or the referring veterinarian with a median follow-up time of 16 months and it was found that surgical treatment was more often associated with clinical improvement compared to medical management.

Long-term prognosis after surgery is difficult to determine because many reports include single case outcomes, use different surgical techniques and have limited follow‐up times. Recurrence of clinical signs after surgery has been reported in a small number of cases (Frykman 1999, Skeen and others 2003, Schmidt and others2007, Sugiyama and Simpson 2009) between 8 and 44 months post-surgery. In only 4 cases (2 dogs and 2 cats) the cause for the deterioration of the clinical signs was investigated and in 3/4 reformation of the SAD was identified on repeated imaging, whereas in the fourth dog MRI did not reveal any abnormalities that could justify the worsening of the clinical signs.

The aims of this retrospective case series were therefore to better characterise the clinical presentation of cases showing late onset neurological deterioration after surgical treatment for SAD and to identify the underlying cause for recurrence of the clinical signs.

**Materials and methods**

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

The medical records of the Small Animal Teaching Hospital of the University of Liverpool, Animal Health Trust and Royal Veterinary College of the University of London were searched retrospectively for patients diagnosed with recurrence of clinical signs after surgery for SAD between January 2010 and June 2016. Patients were included if they had an initial improvement but showed signs of deterioration at least 6 months after surgery and if they underwent repeat MRI to assess the cause for this clinical worsening.

Information regarding signalment, duration of clinical signs, findings of the neurological examination and MRI as well as outcome after initial surgical treatment was collected. Similar data was collected following recurrence of the clinical signs, including findings of the repeat MRI and treatment used at that time along with associated outcome.

MR images of the spine (from both before and after surgery) were reviewed by a board certified radiologist (JFM). The images were acquired using a 1.5T (1.5 T Gyroscan ACS-NT, Philips Medical System) scanner in 2 patients, 1.5T (GE Signa echospeed, GE Milwaukee) in 1 patient and a 1T (Siemens Magnetom, Erlangen, Germany) scanner in 5 patients.

**Results**

Seven dogs and one cat were identified fitting the inclusion criteria (Table 1). All SAD were located in the thoracic and thoracolumbar regions (T2, T8, T9, T11, T12 and T13-L1). Five dogs were male, of which two were neutered. The two female dogs were spayed. The only cat was a neutered male.

Paraparesis was the main clinical sign in all cases. All patients were ambulatory on presentation. Ataxia was present in 6/8 and faecal incontinence in 2/8 cases.

At the initial evaluation, all the SAD were reported to be dorsally located with only one case (no. 8) also comprising a ventral component. All SAD were teardrop shaped; 6 tapered cranially and 2 of them tapered caudally (cases 1 and 8). Only 1 case (no. 3) showed no spinal cord changes relative to the site of maximum compression with the remaining seven revealing intramedullary hyperintensity on T2-weighted images on MRI. The degree of spinal cord compression (evaluated as a ratio on transverse images) ranged between 10% and 64%.

The median age of onset of the initial clinical signs in the canine patients was 36 months (range 16 - 57 months) and the only cat reported was 91 months on presentation. The two Pug dogs in this study presented concurrent neurological disorders, namely a chronic intervertebral disc (IVD) protrusion at the level of the SAD and mild kyphosis; no associated hypoplasia or aplasia of the thoracolumbar caudal articular processes could be appreciated in either case.

Different surgical techniques were carried out depending on the surgeon’s preferences. In cases 1, 6 and 8, debridement of the SAD and durectomy was performed whilst cases 2, 3, 4, 5 and 7 had fenestration, SAD debridement and marsupialisation of the dura mater. The laminectomy defect was covered in cases 1 and 6 with a porcine-derived small intestine submucosa product (SISplus tissue patching material, Avalon Medical, USA) and with a collagen haemostatic fleece (Lyostypt, Braun, Germany) in case 8. Samples from the tissues removed were submitted for histopathology in 4 cases (2, 3, 6 and 7) and analysis revealed a well-organised and dense connective tissue, likely representing the meninges, within which there were small sinus cavities present and in some cases mild inflammatory changes; these findings were compatible with a SAD.

The median follow-up time was 36 months (24 - 68 months). Median time to relapse of the neurological deficits was 20.5 months after surgery (range 9 - 44 months). Six cases represented with deterioration of weakness and pelvic limb ataxia whilst 2 (cases 4 and 8) showed pelvic limb ataxia only. Faecal and/or urinary incontinence occurred in four cases.

On repeat MRI, 3/8 cases showed clear regrowth of the diverticulum (Table 2). In case 1, three MRIs were performed following surgery due to a very slow progression of the clinical signs. After an initial improvement with medical management the dog eventually developed non-ambulatory paraparesis 29 months after the initial surgery; progressive enlargement of the SAD can be seen over this time (Fig 1). Mild to moderate dorsal compression at the previous laminectomy site, which was presumed to be the laminectomy membrane, was identified in 2/8 cases (Fig 2). Herniation of the spinal cord through the laminectomy defect was seen in 3 cases associated with a stellate appearance to the spinal cord with small multiloculated areas of dilation of the subarachnoid space; it was uncertain if these were remnants of the SAD not removed during initial surgery or newly formed (Fig 3). In all eight cases, an intramedullary hyperintensity on T2-weighted images in the region of the previous surgical site was clearly visible on repeat imaging and in 5/6 cases that received contrast medium, mild meningeal enhancement was identified. In one patient with IVD protrusion associated with the initial SAD, there was a mild increase in the degree of compression appreciated on the repeat MRI (case 5).

Various treatment options were elected with subsequent different outcomes (Table 1). In 3 dogs and 1 cat, repeat surgery was performed. This comprised of an extension of the previous laminectomy site, removal of scar tissue, SAD debridement and excision of the overlying dura in cases 1 and 6; in case 1, stabilisation with a SOP plate was also performed at the time of repeat surgery and the T11-T12 IVD fenestrated and the protruded material removed with rongeurs. In cases 7 and 8, removal of scar tissue alone was performed as no evidence of SAD was found on surgical exploration. In all these cases, short courses of corticosteroids were also used.

**Discussion**

Recurrence of neurological signs after surgery for SAD is a rarely described and poorly understood problem in veterinary medicine. This case series shows that different causes can be underlying the clinical signs and that this is not always due to reformation of the SAD. Formation of scar tissue associated with compression of the spinal cord was identified in some cases and herniation of the spinal cord through the laminectomy defect associated with small multiloculated areas of dilation of the subarachnoid space were seen in others. These findings emphasize the importance of repeat imaging in cases with recurrence of clinical signs after surgery for SAD as different underlying causes may warrant different treatment options and carry different associated outcomes.

A recent study in people attempted to provide a new classification for intradural arachnoid diverticula (Klekamp 2017). This divided the arachnoid diverticula into idiopathic or primary (when there was no prior history of an inflammatory accident) and secondary to inflammatory reactions (such as trauma, subarachnoid haemorrhages, meningitis and intradural surgery). Idiopathic cases were significantly more common and had a much lower relapse rate (13% after 1 year and 17% after 5 and 10 years). Patients with secondary diverticula had higher rates of relapse, namely 26% after 1 year; 71% after 5 years and 85% after 10 years. Neurological deterioration after surgery was mostly related to arachnoid scar formation leading to spinal cord tethering, obstruction of CSF flow, and reappearance of a syrinx rather than reformation of an arachnoid cyst. Since arachnoid scar formation tends to form within the first months after surgery, most of the symptomatic relapses occurred within the first 2 postoperative years. Repeat surgery was only performed in a small number of patients but in no case did it result in sustained neurological improvement.

Revision surgery or medical treatment was used in our patients, depending on the imaging findings and pet-owners’ preferences. Half the cases underwent a second surgery and in those in which repeated MRI had revealed SAD recurrence, the clinical signs improved or even resolved (median follow-up time of 34 months). However, in the one case in which no obvious SAD formation had been identified on repeat imaging, surgery resulted in only mild temporary improvement followed by stabilisation of the neurological deficits. These findings support that in cases with recurrence of the SAD as the cause for the neurological deficits, surgery should be considered.

Medical treatment, including anti-inflammatory doses of corticosteroids, physiotherapy, hydrotherapy or a combination of these, was attempted in the remaining 4 cases. Repeat MRI had revealed different abnormalities but none of them was consistent with presence of a clear SAD. Medical treatment resulted in stabilisation of the clinical signs in 2 cases and slow gradual worsening of the clinical signs in the other 2 dogs, eventually resulting in euthanasia. This would suggest that medical management is unlikely to markedly improve the clinical signs in these patients, possibly resulting in poor quality of life and euthanasia.

In 3 cases a clear SAD could be identified and in 3 others, small multiloculated areas of dilation of the subarachnoid space could be seen. As no immediate post-operative MRIs were performed in any case, it is impossible to say if these abnormalities were newly formed or in fact inappropriately removed during the initial surgery. Nonetheless, the fact that there was marked initial improvement if not resolution of the clinical signs in all cases suggests that the latter option is less likely. Also, a clear progression of the SAD could be seen in the case that underwent 3 successive MRIs over a 29-month period after the initial surgery, reinforcing the suspicion that the identified enlargements of the subarachnoid space were in fact newly formed. As no further MRIs were performed in the 3 cases with small dilations of the subarachnoid space and in at least 1 case there was progression of the clinical signs with time, it is possible that these could have become larger and formed well-defined SADs that we failed to identify. Interestingly, the MR images of the three cases with small multiloculated areas of dilation of the subarachnoid space resemble fibrotic pia-arachnoid adhesions (Fisher and others 2013, Meren and others 2017). These lesions remain of unclear aetiology but have been proposed to be secondary to trauma and localised bleeding into the subarachnoid space, which may have occurred during surgery in our patients. It is possible then that in such cases, different surgical techniques such as shunt tube placement should be considered on following surgical interventions (Meren and others 2017).

In 5 cases, the laminectomy defect was not covered before routine wound closure due to the surgeon’s preference. Two of these patients subsequently presented formation of a laminectomy membrane on repeat MRI and the other three showed herniation of the spinal cord into the laminectomy defect. Laminectomy membrane formation is thought to occur during post-operative healing when a haematoma fills the bone defect and this is followed by the development of a fibrous callus, which subsequently undergoes metaplasia to cartilage and bone (Trotter and others 1988). Several techniques have been described in an attempt to reduce the formation of epidural fibrosis in dogs but no agent has been found that consistently achieves this. It is therefore still controversial what the best technique to prevent laminectomy membrane formation is, although it has been suggested that free fat grafts may be associated with a higher rate of neurological complications and should therefore be avoided (da Costa and others 2006). Only one study has previously reported the use of free fat grafts to cover the laminectomy defect in patients undergoing surgery for SAD (Frykman 1999) whilst this is not discussed in any of the others, making it difficult to compare our results with the literature. Nonetheless, our findings suggest that despite lack of sufficient evidence to support the use of a specific agent, covering the laminectomy defect after surgery for SAD is advisable as formation of a laminectomy membrane was only seen in the cases where this was not used.

The 2 Pug dogs in this study presented concurrent spinal disorders, namely IVD protrusions and vertebral abnormalities (wedge-shaped vertebrae and kyphosis). No thoracolumbar articular process hypoplasia or aplasia with secondary constrictive myelopathy was found on the MR images. These congenital vertebral malformations have been reported to cause neurologic deficits difficult to distinguish from other spinal cord diseases, such as intervertebral disc disease, SAD, or neoplasia and can often be difficult to improve despite surgical intervention (Fisher and others 2013). The presence of other spinal abnormalities in these dogs confounds the diagnosis of SAD as the only cause of the clinical signs in these cases. The initial surgical treatment was merely aimed at SAD resolution and both dogs showed marked clinical improvement after surgery. In case 5 nonetheless, the IVD protrusion appeared to be more marked on repeat MRI. No repeat surgery was performed in this case and the clinical signs progressed further, likely due to a combination of the compression secondary to the laminectomy membrane and IVD protrusion.

The lack of histopathological confirmation in half the cases remains an intrinsic limitation of this case series, although the clinical presentation, MR findings, gross intraoperative appearance of the lesions and the post-surgical improvement made other differential diagnoses less likely. Other limitations include the retrospective nature of the study and inclusion of three institutions in which different surgeons used somewhat different surgical techniques.

This case series shows that recurrence of neurological signs after surgery for SAD is not always secondary to diverticulum reformation. In some instances, other causes such as herniation of the spinal cord into the laminectomy defect or laminectomy membrane formation were identified. Therefore, performing advanced imaging is recommended in order to identify the cause of the deterioration and decide on the most suitable treatment options. To the authors’ knowledge, this is the first case series reporting the repeat MRI findings in cases with late recurrence of neurological deficits after surgery for SAD.

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Table 1. Clinical presentation, time to recurrence of clinical signs and outcome of 8 cases with recurrence of clinical signs after surgery for SAD

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Case no.** | **Signalment** | **Clinical signs at initial presentation** | **Outcome after surgery** | **Time to relapse**  | **Clinical signs at recurrence** | **Treatment at reoccurrence and outcome** |
| 1  | 3y 1m M Pug | Paraparesis and ataxia | Improvement in gait although residual paresis and ataxia | 19 m | Worsening of paraparesis and ataxia. Initial stabilisation of signs but became non-ambulatory 29m after initial surgery | Medical treatment with initial improvement. Repeat surgery 29 months after initial surgery resulting in return to ambulation.  |
| 2  | 4y 9m M Shih Tzu | Paraparesis | Improvement in gait although residual paresis and ataxia | 22 m | Worsening of paraparesis, ataxia, urinary and faecal incontinence | Medical treatment. Initial improvement in gait but incontinence persisted. Stable neurological status. |
| 3  | 4y FN Jack Russel Terrier | Paraparesis, ataxia and faecal incontinence | Improvement in gait and resolved faecal incontinence | 34 m | Worsening of paraparesis and faecal incontinence | Medical treatment and hydrotherapy.No improvement but stable neurological status. |
| 4  | 3y FN Shih Tzu | Paraparesis and ataxia | Improvement in gait although residual ataxia | 19 m | Worsening of ataxia | Hydrotherapy. Euthanasia 30 months after surgery due to deterioration. |
| 5  | 1y 4m MN Pug | Paraparesis | Improvement in gait although residual paraparesis | 29 m | Paraparesis, ataxia and faecal incontinence | Physiotherapy.Euthanasia 39 months after surgery due to deterioration. |
| 6  | 7y 7m MN Ragdoll cat | Paraparesis and ataxia | Resolution of the clinical signs | 13 m | Paraparesis and ataxia  | Repeat surgery. Resolution of the clinical signs. |
| 7  | 3y M West Highland White Terrier | Paraparesis, ataxia and faecal incontinence | Improvement in gait although residual ataxia | 44 m | Paraparesis, faecal incontinence. Signs of mild spinal pain. | Repeat surgery. Initial improvement but persistent incontinence and paraparesis. Lost to follow-up 17 months after the second surgery. |
| 8  | 1y 9m M Rhodesian Ridgeback | Tetraparesis and ataxia | Improvement in gait although residual tetraparesis and ataxia | 9 m | Worsening of ataxia | Repeat surgery.Improvement in gait although residual pelvic limb ataxia. |

F – female; M – male; m – month; N – neutered; y – year

Table 2. Findings on initial and repeat magnetic resonance imaging of the 8 patients with recurrence of clinical signs after surgery for SAD

|  |  |  |
| --- | --- | --- |
| **Case no.** | **Findings on initial MRI** | **Findings on repeat MRI(s)** |
| 1 | Dorsal SAD tapering caudally at mid T11. Mild T11-T12 IVD protrusion. Intramedullary hyperintensity on T2WI cranial to SAD. | Reoccurrence of SAD ventrolaterally at mid T11 tapering cranially. Progressive enlargement of SAD over time (3 repeat MRIs). Mild T11-T12 IVD protrusion.Mild meningeal enhancement at surgery site and persistence of intramedullary hyperintensity on T2WI.  |
| 2  | Dorsal SAD at mid T9 tapering cranially. Intramedullary hyperintensity on T2WI caudal to SAD. | Herniation of spinal cord into laminectomy defect with focal meningeal/epidural enhancement. Small focal dilation of subarachnoid space dorsolaterally at surgery site. Extension of intramedullary hyperintensity on T2WI. |
| 3  | Dorsal SAD at mid T9 tapering cranially.  | Mild-moderate dorsal compression of the spinal cord at laminectomy site by probable new bone formation (laminectomy membrane).Development of intramedullary hyperintensity on T2WI at T8-T9. |
| 4  | Dorsal (bi-lobulated) SAD at mid T12 tapering cranially.Intramedullary hyperintensity on T2WI ventral and caudal to SAD | Herniation of spinal cord into laminectomy defect with mild meningeal enhancement. Stellate appearance to the spinal cord with small multiloculated areas of dilation of the subarachnoid space. Persistence of intramedullary hyperintensity on T2WI. |
| 5  | Dorsal SAD at T8 tapering cranially. Mild kyphosis due to wedge vertebrae and mild T7-T8 IVD protrusion. Subtle intramedullary hyperintensity on T2WI ventral and cranial to SAD | Moderate dorsal spinal cord compression by laminectomy membrane. Extension of intramedullary hyperintensity on T2WI. |
| 6  | Bi-lobulated, dorsal SAD at mid T9 tapering cranially. Intramedullary hyperintensity on T2WI caudal to SAD. | Reoccurrence and enlargement of bi-lobulated T9 SAD with more severe spinal cord compression and more extensive caudal spinal cord hyperintensity. Mild herniation of spinal cord into laminectomy defect and mild meningeal enhancement. |
| 7  | Dorsal SAD at T13-L1 tapering cranially. Intramedullary hyperintensity on T2WI caudal to SAD. | Herniation of spinal cord into laminectomy defect with focal meningeal/epidural enhancement. Stellate appearance to the spinal cord with small multiloculated areas of dilation of the subarachnoid space. Persistence of intramedullary hyperintensity on T2WI. |
| 8  | Dorsal and ventral SAD at T2 tapering caudally. Intramedullary hyperintensity on T2WI cranial to SAD.  | Reoccurrence of SAD dorsally and ventrally. Herniation of spinal cord into laminectomy defect.Extension of intramedullary hyperintensity on T2WI. |

IVD – intervertebral disc; L – lumbar vertebra; SAD – spinal arachnoid diverticulum; T – thoracic vertebra; T2WI – T2-weighted images.