Clinical and imaging features of spinal extradural arachnoid cysts: a retrospective study of 50 cases

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Received: 10 May 2022 / Accepted: 11 August 2022 / Published online: 16 August 2022
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Abstract

Purpose  Spinal extradural arachnoid cysts (SEDACs) are thought to arise from leakage of CSF through a spinal dural defect. This study investigates the demographics and imaging spectrum of SEDACs at our academic institution and compares them with those reported in the literature.

Methods  Fifty cases with documented MRI diagnosis of SEDAC, Nabors criteria type I meningeal cyst (MC), were identified from retrospective review of imaging records between 1999 and 2020. Patient demographics, presenting symptoms, cyst characteristics, and management outcomes were studied. Statistical analysis was performed to determine associations between maximum cyst size and presenting symptoms along with other imaging findings.

Results  In all 50 subjects, SEDACs were solitary (single) and sporadic (non-familial). The majority were incidental (62%), located posteriorly (92%) and laterally (80%) in the thoracic and thoracolumbar regions (34%, 30%). They were associated with mild mass effect upon the thecal sac (50%) and bone remodeling (92%). Among symptomatic SEDACs, back pain and radiculopathy were the most reported (68%). Larger cysts were located caudally in the spinal canal, and were associated with greater thecal mass effect, bone remodeling, and septations. Four out of six subjects who underwent surgical management had complete or partial remission. One had cyst recurrence.

Conclusion  In this largest series of SEDACs, most were discovered incidentally, stable over time, and located in the thoracic spine dorsal to the thecal sac. When symptomatic, back pain and radiculopathy were the most common presenting symptoms. Treatment with complete surgical excision may yield the best results for symptomatic lesions.

Keywords  MRI · Spine · Arachnoid cyst · Extradural cyst

Introduction

Arachnoid cysts are pockets of cerebrospinal fluid (CSF) or CSF-like fluid that form next to CSF spaces in the spine or the cranium [1]. Spinal arachnoid cysts are uncommon and can be intradural or extradural. Most of them are spontaneous, while only a few are secondary [1]. Most spinal extradural arachnoid cysts (SEDACs) arise from CSF leaking through a dural defect in the thecal sac and forming an extradural cyst [2]. However, there have also been cases of non-communicating SEDACs where the dural defect was not identified. The etiology of this defect remains unclear, although congenital, traumatic, postsurgical, inflammatory, and infective causes have all been implicated [2]. Genetic etiological factor has been suggested in some reports, including FOXC2 mutations associated with lymphedema-distichiasis syndrome, NRAS gene mutations in neurocutaneous melanosis, and Marfan’s and Milroy’s syndromes [3–6].

Most spinal arachnoid cysts are asymptomatic and are discovered incidentally on MRI or myelography performed for neck or back pain, myelopathy, or radiculopathy [7]. Besides watchful waiting, management of symptomatic cysts

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involves total cyst excision, marsupialization, fenestration, ligation of the communication site, and/or shunting [7].

The classification of spinal arachnoid cysts has always been confusing. In 1988, Nabors et al. [8] proposed a classification to categorize spinal extramedullary meningeal cysts (MC)—a term used interchangeably with arachnoid cysts in the literature—into three types based on spinal nerve presence, spinal level (sacral vs. non-sacral), and dural location (intradural vs. extradural) shown in Table 1 (1). Type IA MC has also been called extradural arachnoid cysts. Nabors et al. also described sacral extradural meningeal cysts (type IB) as “occult sacral meningocele.” In 2017, Klekamp et al. [9] proposed a new classification for spinal dural pathologies similar to vascular aneurysms into saccular dural diverticula (type I), dural dissection (type II), or fusiform ectasia (III) [9–11]. According to this classification, SEDACs will fall under type I diverticula or sometimes type II dissections [9]. Reports of SEDAC in the literature have been limited to small case series, and we sought to expand on this by presenting a retrospective study of 50 cases of SEDAC retrieved from our institution’s health records. To our knowledge, this would be the largest case series of SEDAC in the literature to date.

Methods

Patient identification and selection

To identify patients diagnosed with SEDAC at our institution, a full radiology report record search was conducted using the Illuminate InSight™ clinical search engine. “MRI,” “spinal,” “arachnoid,” “extradural,” and “cyst” were used as search terms. The search yielded 212 reports between the years 1995 and 2021 for 145 patients—some patients had more than one radiology report for different studies. To determine their eligibility, a certified neuroradiologist with over 20-year experience reviewed the patients’ images using Nabors et al. [8] criteria for type I meningeal cysts (extradural cysts). Patients whose imaging was unavailable for review were excluded and a total of 50 patients with a documented MRI diagnosis of SEDAC were identified between 1999 and 2020. All the MRI exams included a minimum of unenhanced axial and sagittal T1-weighted and T2-weighted sequences (1.5 or 3.0 Tesla). All the desired data points were present for all the 50 patients, except two with missing histopathology reports (out of six) and one with a missing operative note (out of six). The study was approved by the institution’s Institutional Review Board (IRB 21–005620–01).

Demographic, clinical, and imaging characteristics

A retrospective chart review for demographic data, presenting clinical symptoms and imaging, surgical, and histopathology findings was conducted. Demographic and clinical data collected were age at diagnosis, sex, date of diagnosis, relevant past medical history (e.g., history of back trauma or surgery), family history, presumed etiology of SEDAC, presenting symptoms, type of management (surgery vs. conservative), date and type of surgery (if any), the presence of subarachnoid communication (from surgical notes and myelography), and pre- and post-operative clinical follow-up. Imaging data collected included SEDAC count, location, size (using largest diameter), seption, thecal mass effect, myelopathy, bone remodeling, and extraforaminal extension. Mean cross-sectional diameter of the thecal sac was used to quantify the degree of mass effect in comparison to adjacent normal-appearing segment. The thecal mass effect was considered mild if it was less than 50%, moderate if between 50 and 75%, and severe if greater than 75% diameter reduction. The presence of subarachnoid communication was recorded when myelography was available. Dates of the first, interval and last imaging follow-up (pre- and post-operative) were also recorded.

Statistical analysis

Continuous variables were reported as medians with interquartile range (IQR) while categorical ones as frequencies. The associations between cyst size, and clinical and imaging findings were assessed using Mann–Whitney (for variables with 2 levels) and Kruskal–Wallis (for variables with 3 or more levels) statistical tests. A p value of less than 0.05 was considered significant. All statistical analysis was performed using SPSS version 27.

Results

General information

A total of 50 patients were included (median age 64 years (IQR 49.2–74.2)). Thirty-one (62%) were female. All 50 patients had no known etiology (i.e., idiopathic), although 5 (10%) patients had an unrelated history of spinal surgery and 2 (4%) had history of unrelated trauma to the

| Table 1 Classification of spinal meningeal cysts by Nabors et al. [8] |
|-----------------|-------------------------------|
| Type | Description |
| I | IA | Extradural meningeal cysts without spinal nerve fibers (extradural arachnoid cyst) |
| | IB | Sacral meningocele (occult sacral meningocele) |
| II | Extradural meningeal cysts with spinal nerve fibers (Tarlov’s perineural cyst) |
| III | Intradural meningeal cysts (intradural arachnoid cysts) |
back. Thirty-two/50 (64%) SEDACs were discovered incidentally while the remainder 18 (36%) presented with symptoms. These were back pain in 10 (56% symptomatic), radiculopathy in 3 (17%), mixed sensory and motor deficit in 3 (17%), and urinary or bowel dysfunction in 2 (11%). Lack of symptoms was noted mainly in relatively cephalad SEDACs with thoracic and thora-columbar cysts being asymptomatic in 12/17 (70%) and 10/15 (67%), respectively, compared to lumbar and lumbosacral cysts in 8/14 (57%) and 2/4 (50%) respectively. Larger SEDACs were significantly associated with symptomatic presentation (47.2 mm (IQR 31.5–99.9) vs. 30.5 mm (IQR 21.5–48.7), \( p = 0.020 \).

**Imaging findings**

Table 2 shows the imaging findings of SEDACs, including cyst count, extraforaminal extension, mass effect upon thecal sac, myelopathy, bone remodeling, septations, location, spinal level along with communication with subarachnoid space, and cyst stability. It also shows the association of SEDAC median diameter to other imaging findings. T6 was the most crossed segment in all thoracic cysts (11/17 cases (65%), followed by T5 (7/17 (41%)) and T7 (6/17 (35%)). There was significant association of larger cysts with extraforaminal extension (\( p = 0.007 \)), thecal sac mass effect (0.001), septations (0.029), and lower spinal regions (0.006).

Of all 50 cases, 25 (50%) had follow-up MRI. The median follow-up duration between diagnosis and the latest MRI was 34 months (IQR 11–95.5). They all showed cyst stability; i.e., no discernible changes in sizes were noted (Fig. 3).

Only seven (14%) subjects were noted to have both an MRI and a CT myelogram performed to evaluate their SEDACs. Of them, 4 SEDACs demonstrated contrast filling, indicating communication to the subarachnoid space. Information regarding subarachnoid communication was also collected from operative notes, which showed positive evidence for another 2 SEDACs with no preoperative myelography. This brought the total number of SEDACs with positive evidence for subarachnoid communication to 6 (66%) among the 9 subjects investigated.

**Management and follow-up**

Forty-four/50 (88%) subjects were treated conservatively with follow-up, while 6/50 (12%) were treated surgically. Five underwent surgical excision, while one had an unknown operation as the operative note was not available. All patients treated with surgery were symptomatic, with three having mixed clinical presentation with both motor and sensory deficits; two having back pain and radiculopathy; and one having urinary and bowel symptoms. Histopathology reports confirmed the diagnosis of an arachnoid cyst in 4/6 (67%) while histology report was unavailable for two patients. All six patients treated with surgery had clinical and MRI follow-up with a median duration between surgery and follow-up of 42 months (IQR 11–66). Postoperatively, 4/6 (67%) had complete or partial remission of preoperative symptoms while the remaining 2/6 (33%) did not respond. Follow-up MRI demonstrated no evidence of residual/recurrent cyst in 5/6 (83%), while 1/7 (17%) had cyst recurrence. One of the two cases without symptom improvement had accompanying lumbar spinal stenosis, likely resulting in residual pain, while the other developed cyst recurrence. The operative note was unavailable for the latter, and it is possible that there was incomplete cyst wall resection or another surgical option (i.e., other than excision, such as cyst fenestration) was sought, which is expected to have a higher chance of recurrence [7].

**Discussion**

Arachnoid cyst is a histological diagnosis. However, in radiology practice, the term is used interchangeably with MC. Despite describing type 1 MC as extradural arachnoid cysts, Nabors et al. identified an arachnoid lining in only one of their six cases and advised against using the term “arachnoid cyst.” However, other studies were able to identify the presence of an arachnoid layer [12, 13]. Nabors et al. also referred to sacral extradural meningeal cysts (type IB MC) as “occult sacral meningocele,” a term seen as inconsistent (with other meningoceles) by Muthukumar et al. [14] because of their similarities to other arachnoid cysts, lack of neural contents, and non-association with spinal dysraphism [14]. Moreover, while Nabors et al. [8] specified the lack of spinal nerves within the cyst to characterize SEDACs in contrast to perineural (Tarlov’s) cysts, the herniation of spinal nerve roots through dural defects has been reported in SEDACs before [15, 16]. Differentiating between sacral SEDACs and Tarlov cysts can sometimes prove difficult. This study considered cysts centrally placed in the sacral canal and extending over multiple segments as sacral SEDACs. In contrast, those eccentrically located at a single sacral level, extending into a sacral neural foramina and containing nerve elements, were excluded as Tarlov’s cysts [17].

MRI constitutes the primary imaging modality for diagnosis. On MRI, SEDACs exhibit T1-weighted hypointense and T2-weighted hyperintense CSF-like signal intensity [17, 18]. High-resolution MRI sequences, including constructive interference in steady-state (CISS) and dynamic cardiac gated cine-mode balanced steady-state free precession imaging (SSFP), have been suggested as a helpful non-invasive tool for evaluating possible communication to subarachnoid
This could help identify the target surgical site and limit the extent of laminectomy required [1]. Moreover, kinematic MRI can help correlate clinical symptoms with the cyst shape fluctuation that happens with daily activities, e.g., straining [20]. On the other hand, in the absence of bone remodeling, CT can easily miss SEDACs as they appear isodense to CSF [13]. However, it is reliable in elucidating related bone abnormalities, and when performed with myelography, can readily distinguish between communicating and non-communicating SEDACs (Figs. 1 and 2) [18, 21]. Nearly all SEDACs, based on literature, communicate with the subarachnoid space [22]. Digital substraction cystography can reveal the site of communication of SEDAC with the thecal sac [23].

### Table 2 Imaging findings of SEDACs among study subjects (n = 50) and their association to cyst median highest diameter

| Imaging findings                        | Number of cases (% among total) | Median diameter (IQR) (mm) | p value† |
|----------------------------------------|----------------------------------|---------------------------|---------|
| Number of cysts                        | Solitary                         | 50 (100%)                 | 36.3 (22.8–56.7) | - |
|                                        | Multiple                         | 0 (0%)                    | -       |
| Extraforaminal extension               | Present                          | 41 (82%)                  | 40.1 (26.8–65.3) | 0.007 |
|                                        | None                             | 9 (18%)                   | 22.4 (14.9–29)   |          |
| Thecal mass effect                     | None                             | 11 (22%)                  | 26.4 (20–33)     | Less than 0.001 |
|                                        | Mild                             | 25 (50%)                  | 31 (21.7–44.6)   |          |
|                                        | Moderate                         | 7 (14%)                   | 52.4 (40–99.8)   |          |
|                                        | Severe                           | 7 (14%)                   | 100.1 (72–112)   |          |
| Myelopathy                             | Present                          | 2 (4%)                    | 71         | 0.14    |
|                                        | None                             | 40 (80%)                  | 31         |          |
|                                        | Not applicable*                  | 8 (16%)                   | -         |          |
| Bone remodeling                        | Present                          | 46 (92%)                  | 38.3 (22.8–56.7) | 0.508 |
|                                        | None                             | 8% (4)                    | 29 (16.5–62.5)   |          |
| Loculation/septations                  | Present                          | 31 (62%)                  | 42 (27–65)      | 0.029   |
|                                        | None                             | 19 (38%)                  | 25.3 (20–42.7)   |          |
| Location                               | Anterior                         | 4 (8%)                    | 50.8 (33.9–65.4) | 0.334   |
|                                        | Posterior                        | 46 (92%)                  | 33.7 (22.3–52.7) |          |
|                                        | Central                          | 10 (20%)                  | 49.8 (25.1–99.8) | 0.452   |
|                                        | Lateral                          | 40 (80%)                  | 36.3 (22.5–50)   |          |
| Region/level                           | Cervical/cervicothoracic         | 0 (0)                     | -         | 0.006   |
|                                        | Thoracic                         | 17 (34%)                  | 25 (18.5–33.5)   |          |
|                                        | Thoracolumbar                    | 15 (30%)                  | 50 (31–75)      |          |
|                                        | Lumbar                           | 14 (28%)                  | 36.5 (22.3–44.7) |          |
|                                        | Lumbosacral                      | 2 (4%)                    | 108.5       |          |
|                                        | Sacral                           | 2 (4%)                    | 92.8        |          |
| Other findings                         | None                             | 44 (88%)                  | 36.5 (23.5–53.5) | 0.383   |
|                                        | Tarlov cysts                     | 3 (6%)                    | 36.6        |          |
|                                        | Cranial Arachnoid cysts          | 2 (4%)                    | 42.5        |          |
|                                        | Syringomyelia                    | 1 (2%)                    | 17          |          |
| Communication with subarachnoid space  | Present                          | 6 (12%)                   | 51.2 (33–99.8)  | 0.739   |
|                                        | None                             | 3 (6%)                    | 40          |          |
|                                        | Not applicable**                 | 41 (82%)                  | -          |          |
| Stability                               | Unchanged/stable                 | 25 (50%)                  | 31.4        |          |
|                                        | Decreased in size                | 0 (0)                     | -          |          |
|                                        | Increased in size                | 0 (0)                     | -          |          |

†Mann–Whitney U, and Kruskal–Wallis H tests were used

*The cyst was located inferior to the spinal cord

**Subjects had no prior myelography, nor was there any mention of subarachnoid communication/non-communication on the operative notes

‡Only 50% (25) of the subjects had follow-up MRIs. The median duration was 34 (11–95.5) months

Bold entries are “statistically significant” i.e. p value of less than 0.05
Spinal arachnoid cysts usually present in the third or fourth decade of life [24]. The median age of subjects in this study was 64 years (IQR 49.2–74.2). Female predominance (31:19) in this study is concordant to some studies [18, 25, 26] and contrary to others [18, 25, 26]. All 50 subjects with SEDACs were found to have solitary (single) cysts. Although neither of the study subjects (nor their families) underwent thorough genetic analysis, the mere lack of family history and the cyst singularity point to them as non-syndromic. In Ogura et al. [3], 17 subjects with SEDACs were studied, and they found that 7 out of 7 (100%) subjects without FOXC2 mutations had a single SEDAC, whereas 7 out of 10 (70%) subjects with FOXC2 mutations had multiple SEDACs. However, familial SEDACs could, in theory, be easily missed because they may lack symptoms or even have incomplete penetrance, i.e., possess the culprit mutation(s) without SEDAC development. Other associated findings included a thoracic spinal cord syrinx in one subject. In contrast to other studies which postulated causative relation between syringomyelia with spinal and/or cranial arachnoid cysts [13, 27], in our case, the syringomyelia appeared unrelated and incidental as it was caudal and distant from SEDAC.

The majority of SEDACs are found incidentally [17]. Patients may remain asymptomatic until sufficient expansion of the arachnoid cyst compresses the neural elements [28]. Additionally, the location of the cyst perhaps influences the clinical presentation [29]. Whenever a SEDAC is discovered in a symptomatic patient, it must be determined if there is a better explanation for symptoms and whether the cyst can cause the symptoms. SEDAC found with an accompanying lesion, e.g., disk herniation, may pose a diagnostic dilemma [30]. Therefore, clinical presentation must match the radiographic results if treatment is to be successful [14, 17].

Larger cysts were significantly associated with higher degree of thecal mass effect and extraforaminal extension. Postulated mechanisms for cyst enlargement include (a) a one-way valve with intermittent high pressure, (b) hyperosmolar fluid collection causing free water movement into the cyst, and (c) fluid secretion from the cyst wall lining in the absence of communication with subarachnoid space [31]. For nonsurgical patients who had follow-up MRIs, all

Fig. 1  A Sagittal T2W MRI, B sagittal CT myelogram, C axial CT myelogram, and D axial T2W MRI show a posterior thoracolumbar SEDAC at T11-L2 (white arrows). It is seen as a high signal intensity extradural lesion on MRI. CT myelogram images show opacification of the cyst consistent with communication to the subarachnoid space. Note the compression of the thecal sac (severe mass effect) and anterior–posterior widening of the spinal canal due to remodeling by the cyst. The remodeling is best appreciated on the sagittal images as smooth posterior scalloping of the vertebral bodies and erosion of the spinolaminar junctions at the level of the cyst (black arrowheads). Also, note the thin septum within the cyst (white arrowhead).
cysts remained stable in size and appearance (Fig. 3). Pressure changes caused by Valsalva maneuver–related daily activities have been postulated to fluctuate cyst size and precipitate symptoms intermittently [14, 20]. There was also a relative size discrepancy between SEDACs associated with bone remodeling suggestive of long-standing process versus those without bony change [20]. Not surprisingly, larger cysts with higher hydrostatic pressure are expected to gradually remodel the surrounding bone [30]. Also, it is worth noting that bony changes can happen long before the appearance of signs of spinal cord compression [32], which would explain why the majority (29 out of 46 [63%]) of bone remodeling-SEDACs were discovered incidentally.

SEDACs are most common in the mid to low thoracic spine and are usually in posterior location within the spinal canal [26]. The T6 followed by T5 and T4 vertebrae were the most common segments crossed by thoracic SEDACs. Sadek et al. [33] reported T5 to be the most frequent level, followed by T3, T4, and T6. The largest

Fig. 2  A Sagittal T2W MRI, B sagittal CT myelogram, C coronal T2W MRI, and D coronal CT myelogram show a large sacral SEDAC (white arrows) producing marked scalloped enlargement of the sacral spinal canal (black arrowheads). The cyst’s lack of opacification on CT myelogram favors non-communication to subarachnoid space

Fig. 3  A, B Sagittal T2W MRI follow-ups of an incidental stable thoracic SEDAC (white arrows) in 2002 and 2003, respectively
SEDACs were seen in the lumbosacral and the sacral regions where they were most symptomatic. The small median size of the more cephalad SEDACs could explain that group's relative lack of symptoms. Although it cannot be substantiated, some attribute the common posterior location of SEDACs to diverticula in the septum posticum, a thin longitudinal membranous partition that divides the posterior spinal subarachnoid [7, 32]. This does not explain the pathogenesis of the ventral SEDACs. Also, it does not explain the presence of sacral SEDACs where septum posticum is not found [33]. Another theory by Fortuna et al. might help explain the mechanism of ventrally located SEDACs [12]. They suggested that herniation of an entrapped dilated arachnoid granulation through a dural tear it produces, facilitated by negative epidural pressure, leads to their expansion along the path of least resistance, and creation of SEDAC, while the tendency of SEDACs for lateral location can be attributed to common occurrence of tears at the nerve root dural sleeve—thecal sac junction [12].

Conclusion

SEDACs are uncommon entities. MRI presents a valuable tool for both diagnosis and follow-up. The findings of this most extensive compilation of SEDAC cases to date (50 cases) are consistent with prior literature. Most SEDACs were discovered incidentally, stable over time, and most frequently located in the thoracic region, dorsal to the thecal sac. Back pain and radiculopathy constituted most of the presenting symptoms. The larger the cyst size, the greater was the mass effect on the thecal sac, bone remodeling, and chances of internal septations. The caudal levels had a preponderance of bigger cysts, with the largest seen in the lumbosacral and the sacral regions.

Declarations

Conflict interests We declare that we have no conflict of interest.

Ethics approval IRB exempt study.

Informed consent Not applicable.

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