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Research article

Spinal extradural arachnoid cysts: A novel formation mechanism and dural defect location technology

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ABSTRACT

Purpose: The formation mechanism of spinal extradural arachnoid cysts (SEACs) remains unclear. There are several hypotheses for the formation of SEACs, but none of them can fully explain its pathological findings and surgical procedures. In this study, we retrospectively analyzed the cases of SEACs, aiming to clarify the formation mechanism of SEACs. In addition, we summarize a concise method for locating dural defects preoperatively and formulate a putative explanation of this method.

Methods: The clinical data of 14 patients with SEACs underwent surgery in our hospital from January 2017 to December 2021 were retrospectively analyzed.

Results: Fourteen patients were identified during the study period. The cysts all spanned the T12/L1 segment, and dural defects were also located at the T12/L1 level (2 cases not recorded) as well as the middle or the upper-middle level of the cysts. Nine cases were treated with total cyst excision, 2 cases were treated with dural defect closure only, and 3 cases were treated with total cyst excision and dural defect closure. Histopathological examination demonstrated that the cyst wall contained both the arachnoid epithelial and compact fibrous connective tissue. The symptoms were relieved in all patients, and no recurrence was observed.

Conclusions: According to intraoperative and pathological findings, the dural outer layer cyst (DOLC) is a more reasonable hypothesis about SEACs formation. When CT myelography or cinematic MRI cannot determine the location of the dural defect preoperatively, it can be located according to the middle level of the SEACs with high accuracy.

1. Introduction

Spinal extradural arachnoid cysts (SEACs) are rare spinal space-occupying lesions, usually found dorsally to the dural sac at the thoracolumbar junction [1,2]. The reasons for this distribution include that the thoracolumbar junction is the transition zone of mechanical stress from the stable thoracic spine to the flexible lumbar spine, and the local dural is more likely to be damaged [3]. In addition, the hydrostatic pressure in the subarachnoid space is higher toward the caudal end of the spine.

The pathogenesis of SEACs is not fully understood. It is generally believed to be diverticula of the arachnoid or dural mater due to a

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dural defect caused by congenital or acquired factors (trauma, inflammation, hemorrhage, iatrogenic). The valve-like mechanism between the cyst and subarachnoid space may play an essential role in the enlargement of SEACs, which allows CSF to move intermittently from the subarachnoid space to the cyst [4,5]. The fold of the meninges or nerve root fiber at the cyst's ostium may act as the one-way valve [3,6].

The formation mechanism of spinal extradural arachnoid cysts (SEACs) remains unclear. There are several hypotheses for the formation of SEACs, including extradural arachnoid cysts, dural diverticulum cysts, and dural dissection cysts. Still, none of them can fully explain its pathological findings and surgical procedures. In this study, we retrospectively analyzed the cases of SEACs, aiming to clarify the formation mechanism of SEACs. In addition, we summarize a concise method for locating dural defects preoperatively and formulate a putative explanation of this method.

2. Methods

We searched medical records for all patients with a diagnosis of SEACs underwent surgery by the spine surgery professional team composed of 4 surgeons in our hospital from January 2017 to December 2021. All patients with pathology reports of SEACs were included. We excluded other spine cystic disorders, such as synovial cysts, intradural cysts and cystic tumors. Patients with recurrent or previously treated SEACs were also excluded. All patients underwent surgical procedures with total cyst excision or dural defect closure. Dural defects were located according to the middle level of the SEACs by preoperative sagittal T2-weighted magnetic resonance imaging (MRI). The demographics, preoperative and postoperative symptoms, duration of symptoms, preoperative and

Table 1
Clinical Features, imaging findings, surgical procedures, and outcomes for patients included in the present study.

No	Age (y) /Sex	Symptoms	SD (y)	Level	MLS	Defect Position	Sides	Multiple Cysts	Surgical Methods	FU (m)	CRD	CRR	SP (%)
1	63/F	back pain	1	T12-L3	L1/2	L1	left	no	L1-2 laminoplasty excision	49	no	no	90
2	49/F	back and left leg pain	2	T12-L3	L1/2	L1	left	no	T12-L3 laminoplasty excision	42	yes	no	90
3	46/F	Bilateral leg numbness	5	T11-L3	L1	T12	left	no	T12-L2 laminoplasty excision	28	no	no	70
4	26/M	bilateral leg numbness	4	T9-L5	–	NR	NR	yes	T9-12 right laminectomy L1-4 laminoplasty excision	26	no	no	70
5	14/M	right foot drop	1	T12-L2	L1	L1	left	no	T12-L2 laminoplasty excision + suture	20	no	no	50
6	67/M	bilateral leg numbness	2	T11-L1	T12	T12	left	no	T11-L1 laminoplasty excision	19	no	no	70
7	43/M	bilateral leg weakness	25	T8-L2	–	T11/T12	left	yes	T9-T12 laminoplasty suture	18	no	no	70
8	13/M	back pain	1	T11-L1	T12	T12	right	no	T11-12 laminoplasty excision	18	no	no	80
9	41/F	back left leg pain	1	T10-L3	T12/L1	T12	left	no	T11-L2 laminoplasty excision	17	no	no	90
10	58/F	bilateral leg weakness	40	T2-L4	–	NR	NR	yes	T4-5, T8-9, L1, L3 laminoplasty excision	14	no	no	60
11	45/M	left leg numbness	1 (m)	T10-L2	T12	L1	left	no	T11-L2 laminoplasty excision + suture	14	no	no	80
12	21/F	foot dorsiflexion disability	5	T10-L2	T12	T12	right	no	T11-L1 laminoplasty excision	12	yes	no	70
13	33/F	right leg pain	3	T12-L1	T12/L1	T12	right	no	T12-L1 right laminectomy excision + suture	9	no	no	90
14	65/M	left leg numbness and weakness	3	T11-L1	T12	T12	left	no	T12 laminoplasty suture	7	no	no	90

Abbreviation: SD= Symptoms Duration; MLS = Middle Level of the SEACs; FU= Follow Up; CRD=Cyst Residual; CRR=Cyst Recurrence; NR = not recorded; SP=Symptom improvement; y = years; m = months.

postoperative MRI, intraoperative findings, the cyst segment, the location of the dural defect preoperative and intraoperative, single or multiple cysts, cyst residual, and cyst recurrence were reviewed and analyzed. We evaluated the accuracy of our localization method according to whether the dural defect located preoperatively is consistent with intraoperative findings. In addition to preoperative and postoperative clinical evaluation, follow-up MRIs were performed three days, three months, and one year after surgery. The improvement of symptoms after surgery is quantified by the patient's subjective statement and presented in percentage. All patients signed surgical consent forms preoperatively.

2.1. Statistical analysis

Data are expressed as mean ± standard deviation or median (25th–75th percentile). The normality of variables was assessed. All statistical analyses were performed with IBM SPSS Statistics 25.

3. Results

Fourteen patients were identified during the study period, which accounted for about 0.92% of 1520 cases of spinal space-occupying lesions during the same period, with a male to female ratio of 1:1 and an age range of 13–67 years (41.7 ± 4.9 years). The chief complaints were pain (5 patients, 35.7%), numbness (5 patients, 35.7%), and weakness of the lower limb (4 patients, 28.6%). The duration of symptoms varied widely, ranging from 1 month to 40 years, with a median duration of 2.5 years (1.0–5.0 years). In our series, only one patient had a history of trauma. None of the patients had a history of inflammation, hemorrhage, and spine surgery. The cysts all spanned the thoracolumbar junction with a median of 4.5 segments (3–6.25 segments), and dural defects were also located at the T12/L1 level (2 cases not recorded) as well as the middle or the upper-middle level of the cysts. The dural defect is located on the left side in 9 cases and the right in 3 cases. Nine cases were treated with total cyst excision; two of the cases had residual cyst after the operation, but without dural sac compression, and the symptoms were relieved. 2 cases were treated with dural defect closure only, and 3 cases were treated with total cyst excision and dural defect closure. There were 3 cases of multiple cysts, of which 2 cases underwent total cyst excision, and 1 case underwent dural defect closure. Histopathological examination demonstrated that the cyst wall contained both the arachnoid epithelial and compact fibrous connective tissue. The symptoms improved in all patients, and

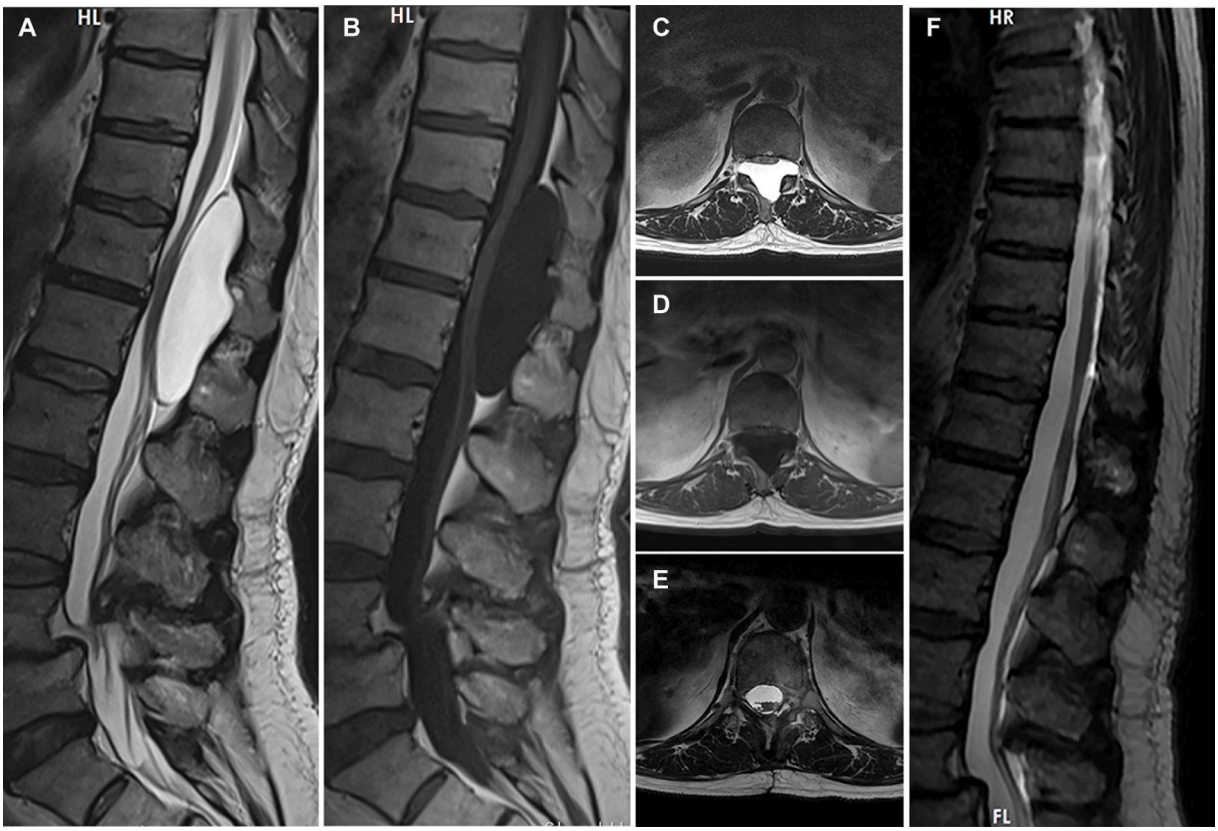

Fig. 1. Preoperative and postoperative magnetic resonance imaging. Preoperative T2-weighted sagittal and axial images demonstrate an epidural cyst from T11 to L1, situated dorsal to dural sac extending to the foramen (A and C). Preoperative T1-weighted sagittal and axial images (B and D). Postoperative T2-weighted sagittal and axial images show the cyst disappeared, and the dural sac compression is relieved (E and F).

no recurrence was observed. One patient developed a cerebrospinal fluid leak. All data are outlined in Table 1. Written informed consent was obtained from the patients to publish their cases and images, and this study was ethically approved by the ethics committee of Xiangya Hospital, Central South University.

3.1. Case illustrations

3.1.1. Case 1

A 65-year-old man was admitted to the hospital for numbness and weakness of the left lower limb for three years. Neurological examination showed that the left lower limb muscle strength was grade Ⅳ, and the superficial sense of the left lower limb was decreased. The preoperative magnetic resonance imaging (MRI) demonstrated an epidural lesion extending from T11 to L1 with a low-

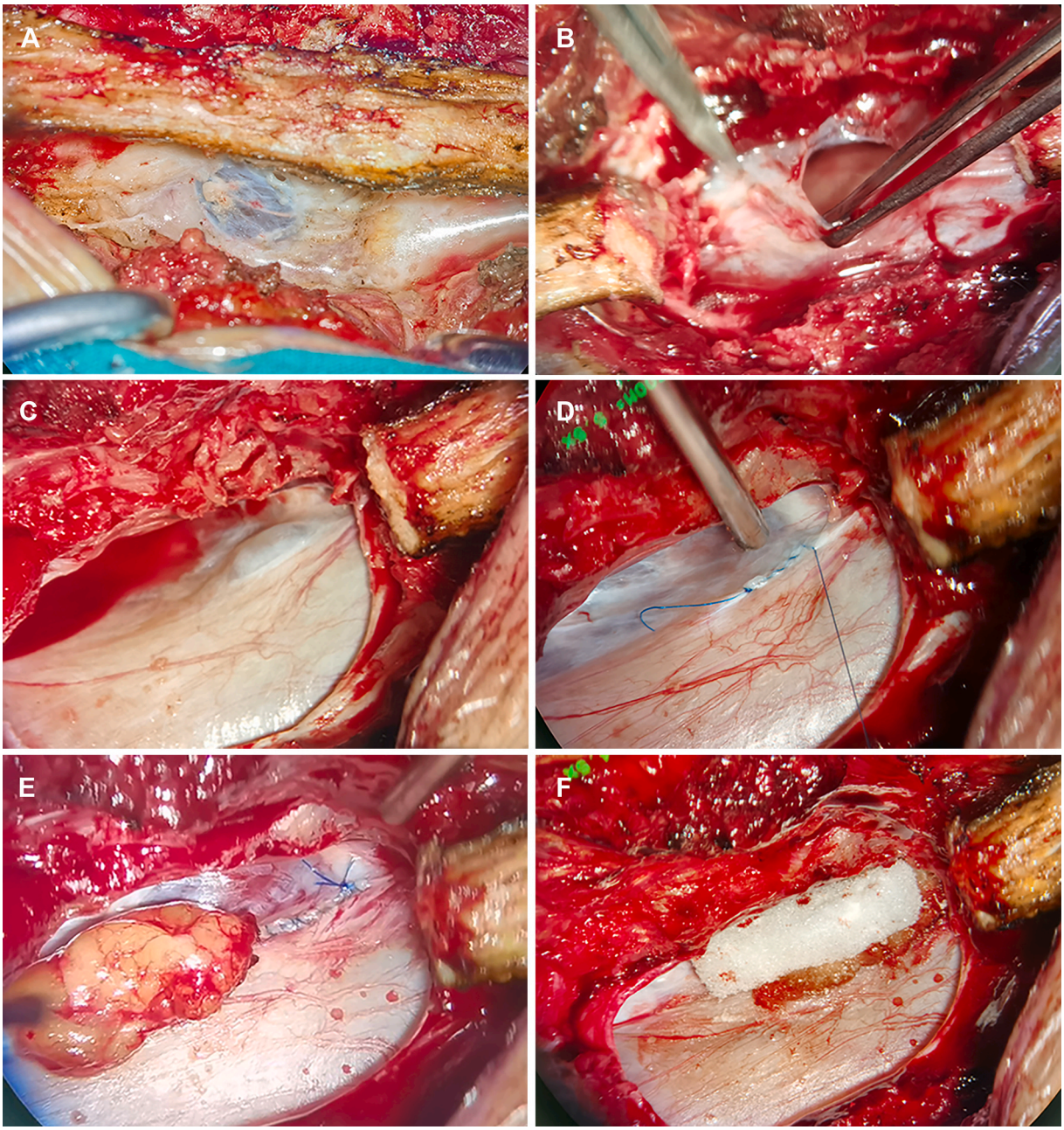

Fig. 2. Repair of dural defect under the microscope. Exposure of the spinous processes and lamina (A). Resection of the dorsal cyst wall (B). Exposure of the defect (C). 7-0 PROLENE was used to suture the defect (D). Autogenous adipose tissue and artificial bio-glue were used to cover and seal the defect (E and F).

intensity signal on T1-weighted and a high-intensity signal on T2-weighted with enlarging of the spinal canal, destruction of the lamina, and compression of the dural sac. (Fig. 1A, B, C and D).

According to our localization method, we judged that the dural defect might be at the T12 level (middle level from T11 to L1), so we performed single-level laminoplasty of T12 and closed the dural defect without excision of the cyst. During the operation, we found the dorsal wall of the cyst was translucent, and the local lamina became very thin under long-term hydrostatic pressure (Fig. 2A). After resectioning the dorsal cyst wall, the dural defect was visualized on the left side of the upper border of the T12 level. A dural defect with sharp edges is covered by an arachnoid membrane that may act as a one-way valve. Local pulsation and cerebrospinal fluid flow can be seen in the defect (Fig. 2B and C). After suturing the defect with 7-0 PROLENE, autogenous adipose and artificial bio-glue were used to cover the defect to ensure water tightness (Fig. 2D, E, and F). Finally, pulmonary pressure was elevated by the ventilator (Valsalva maneuver) to confirm the watertight suture.

MRI revealed that the cyst disappeared three months after the operation, and the patient's symptoms improved by about 90% (Fig. 1E and F). No postoperative complications.

3.1.2. Case 2

A 58-year-old woman presented with bilateral lower limb weakness for 40 years. Neurological examination demonstrated grade 3 muscle strength in both lower extremities, with tendon hyperreflexia and positive Babinski sign. MRI showed several cystic lesions separated from each other from T2 to the caudal canal, which were suspected to be syndromic SEACs. They had slightly higher intensity than cerebrospinal fluid (CSF) on T1 and T2 weighted images. The differential diagnosis of cystic lesions needs to consider the possibility of tumors, such as schwannoma. However, an enhanced MRI showed no enhancement of the cystic wall. Considering that the cyst was located in the epidural, the intradural cyst was also excluded (Fig. 3A and B).

Given multiple cysts and defects, it is difficult to locate all defects preoperatively; total cyst excision was performed. To fully expose every cyst, we performed T4-5, T8-9, L1, and L3 laminoplasty during the operation. A total of four epidural cysts were found during the operation, respectively located in the T2-4, T5-8, T9-L1, and L2-4. Every cyst wall was separated from the dura mater to expose their caudal and rostral boundaries. The cysts were totally excised, and no defect repair was performed. During the process of cyst dissection, the gelatin sponge was used to control the bleeding from the epidural venous plexus. Long segment laminectomy may lead to more severe kyphosis and segmental instability [7], so we performed laminoplasty using a small titanium plate and screw fixation

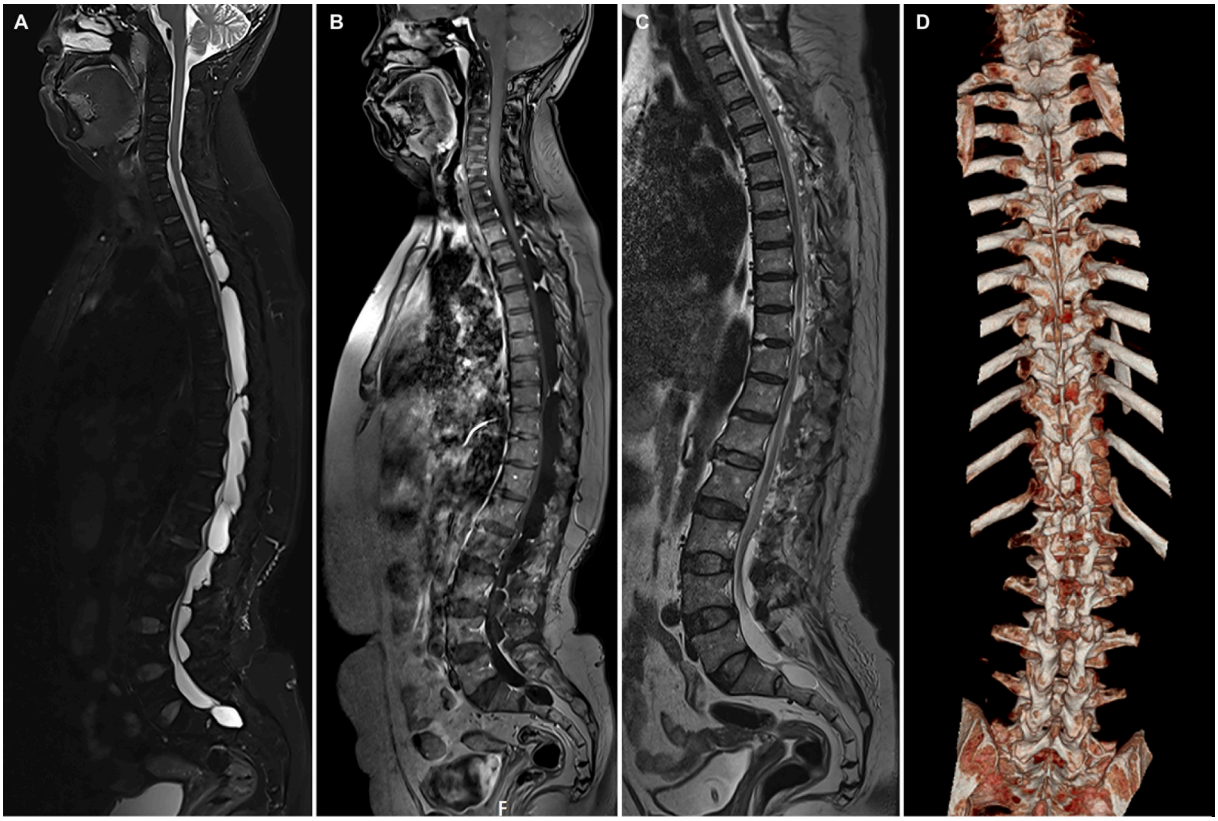

Fig. 3. Preoperative and postoperative imaging. Preoperative T2-weighted sagittal image demonstrates several cystic lesions from T2 to the caudal canal (A). Preoperative enhanced T1-weighted sagittal image (B). Postoperative T2-weighted sagittal image shows complete decompression of the dural sac (C). Postoperative three-dimensional computed tomography shows laminoplasty using a small titanium plate and screw fixation (D).

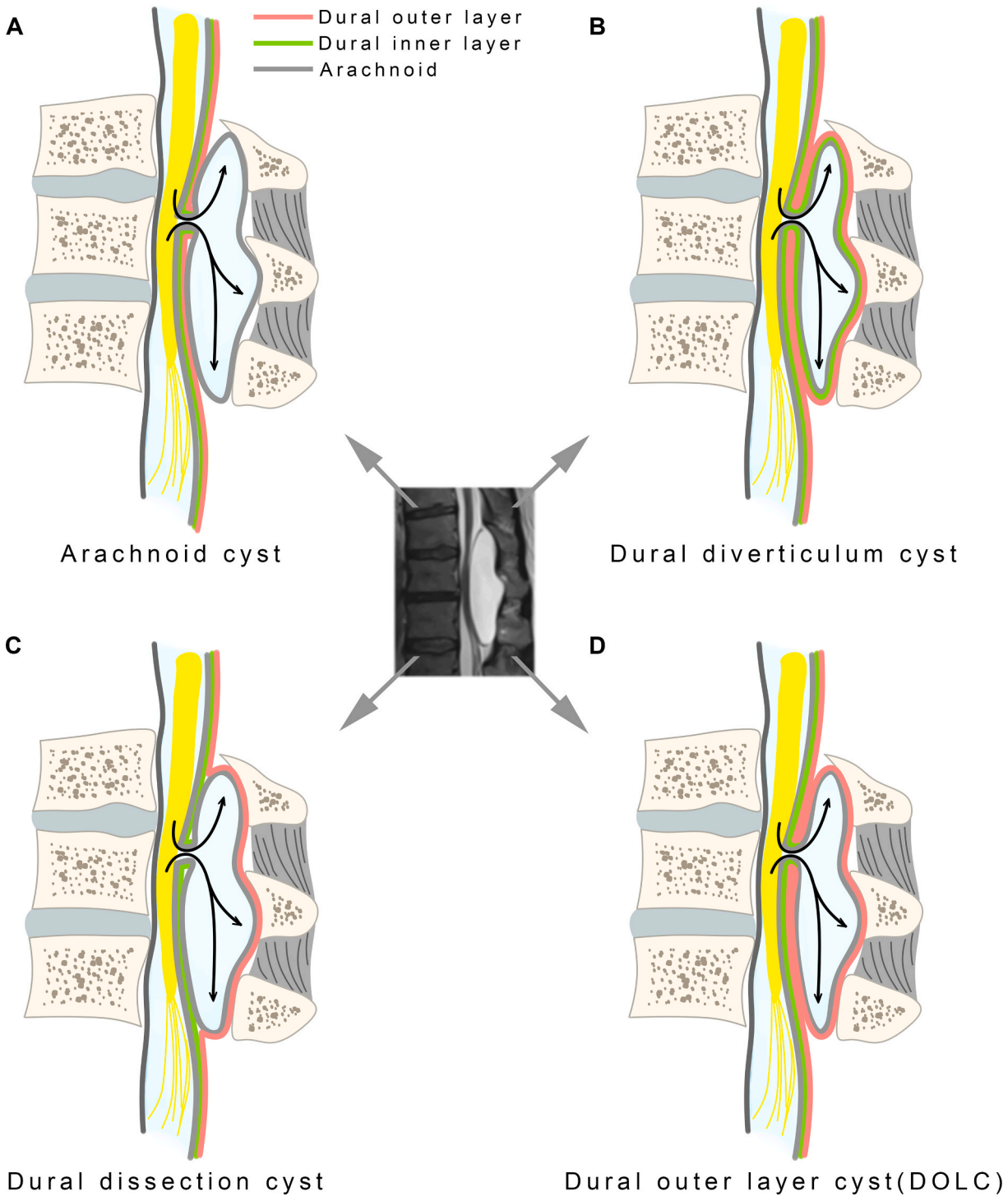

Fig. 4. The formation mechanism of SEACs. Arachnoid cyst hypothesis. The arachnoid herniates through the dural defect (A). Dural diverticulum cyst hypothesis. The cyst is a dural diverticulum (B). Dural dissection cyst hypothesis. The CSF accumulates between the dural inner and outer layers through an inner layer fistula (C). Dural outer layer cyst (DOLC) hypothesis. When congenital or acquired factors break a small cleft on the dural inner layer with an intact outer layer, the hydrostatic pressure and valve-like mechanism with the unidirectional flow of CSF cause the outer layer to gradually expand (D). The red line represents the dural outer layer; the green line represents the dural inner layer; the gray line represents the arachnoid. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

(Fig. 3D). The patient's symptoms improved by about 60%. Postoperative MRI showed that the cysts disappeared, and the dural sac compression was relieved (Fig. 3C). No postoperative complications.

4. Discussion

4.1. Surgical treatment

The standard treatment for SEACs is total cyst resection and closure of communication between the cyst and the arachnoid space. Its

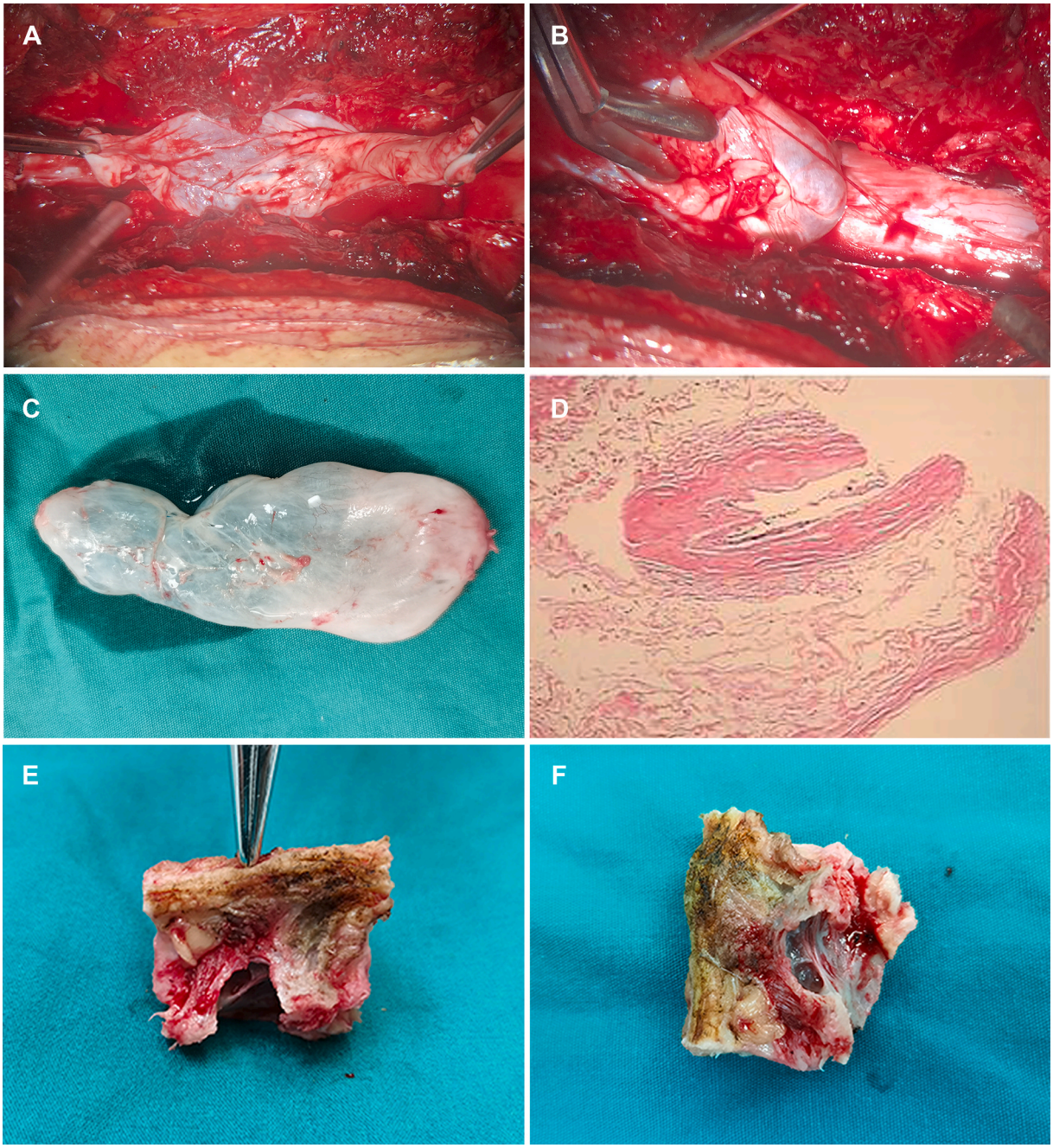

Fig. 5. Pathological and intraoperative findings. The cyst could be removed en bloc after wholly separating from the dura (A and B). Injecting water into the excised cyst can form an independent water pocket (C). The cyst wall contained the arachnoid epithelial and compact fibrous connective tissue (D). Bone erosion at the dural defect site (E and F).

optimal surgical treatment remains controversial [7–9]. Several other surgical treatment methods have been reported: cyst fenestration, cysto-peritoneal shunt, and subarachnoid-cyst shunt, which had a higher chance of recurrence [10-12]. In some cases, the defect could not be found, or the dura around the defect was too thin to be sutured. Total cyst resection seems more necessary than repairing the dural defect [13]. Hatashita et al. believed that the closure of the defect between the cyst and the subarachnoid space is unimportant. If the cyst is completely excised, even if the defect is not closed, the cyst will rarely recur after surgery [14]. This view was also confirmed in our cases.

However, in the case of long-segment cysts, extensive laminectomy or laminoplasty is required to expose the total cyst, which has an adverse effect on the spine's stability and is prone to complications such as increased bleeding, muscle damage, postoperative kyphosis, and scoliosis [7,15]. Recent studies have also demonstrated that the recurrence of the SEACs was related to the closure of the dural defect rather than complete cyst excision. Cyst recurrence rates of 2% and 10% in patients with and without fistula repair, respectively [16]. By comparing the two different surgical methods: total cyst excision and dural defect closure, Funao et al. believe that there was no significant difference in postoperative neurological recovery between the two surgical methods, but dural defect closure without cyst resection via selective laminectomy was less invasive and thus prevented postoperative kyphosis [7]. A growing body of research supports the above study [17–19]. Some scholars locate the dural defect by the size of the left or right laterality of the cyst and then use the hemilaminectomy [18,19]. However, for most SEACs, because the cyst is large and symmetrical in the spinal canal, it is difficult to judge which side the dural defect is located on by preoperative imaging. In addition, regardless of total cyst resection or closure of the dural defect, the hemilaminectomy is limited in exposure. Therefore, we usually adopt laminoplasty to avoid delayed kyphosis seen after laminectomy.

In our study, the patient's symptoms improved regardless of surgical methods. Although there were two patients with residual cysts after the operation, no recurrence was found. During the course of surgical treatment, no severe complications were noted; only one patient developed a cerebrospinal fluid leak after the operation. We believe that both surgical methods have satisfactory outcomes. However, dural defect closure was preferred over total cyst excision because of fewer spinal segment exposure and minor surgical trauma.

4.2. Cyst formation mechanism and factors contributing to cyst enlargement

The exact formation mechanism of SEACs is unclear. It is argued that the arachnoid membrane herniates through a minor dural defect and expands to form an extradural cyst under the action of hydrostatic pressure [8,20–23] (Fig. 4A). The fold of the meninges or nerve root fiber at the cyst defect maybe act as a one-way valve, allowing CSF to become trapped within the cyst [3,6]. Furthermore, the residual arachnoid secreted CSF may also play a role in cyst expansion [24,25].

However, Histopathological examination demonstrated that the cyst wall contained both arachnoid mater and dura mater [12,13, 18,19,26], consistent with our pathological results, the arachnoid epithelial and compact fibrous connective tissue (Fig. 5D), which indicates that the cyst is an outer pocket formed by dura mater, rather than a simple arachnoid cyst.

Based on the pathological findings, some have proposed that the cyst is a dural diverticulum via a defect involving both dural layers, which is enlarged by CSF (Fig. 4B) [18,26]. In contrast, we found that the defect is usually a straight fissure with a sharp edge, which cannot be formed by the dural diverticulum (Fig. 2C). The dural diverticulum cyst hypothesis seems to be unreasonable.

Hamburger et al. consider that dural cysts originate from splitting the two leaves of the spinal dural mater with CSF accumulation, and the adherence of the two leaves is significantly reduced [27]. Chen et al. put forward the dural dissection cyst (DDC) hypothesis based on intraoperative findings (Fig. 4C). The mechanism for DDC formation is that CSF accumulates between the dura inner and outer layers through an inner layer fistula, just like the dissecting aneurysm formation [12].

However, it was always found that the SEACs had a pedicle linking with dura mater [4,28]. In our cases of total cyst excision, most cysts were round on both blind ends, and the cysts could be removed en bloc after wholly separating from the dura [9,13,28,29] (Fig. 5A and B). Injecting water into the excised cyst can form an independent water pocket (Fig. 5C). The dural dissection cyst hypothesis is difficult to apply to explain the above intraoperative finding.

Based on the pathological and intraoperative findings, we proposed the dural outer layer cyst (DOLC) hypothesis. As shown in Fig. 4D, when congenital or acquired factors break a small cleft on the dural inner layer with an intact outer layer, the hydrostatic pressure and valve-like mechanism with the unidirectional flow of CSF cause the outer layer to gradually expand, and the arachnoid can herniate along the cleft into the cyst. The expanded dural outer layer cyst compresses the dural sac and nerve roots, causing various symptoms.

4.3. Location of dural defect

In our cases, the dural defect was usually in the axilla of the nerve root, probably due to the tension between the movable dural sac and the fixed nerve root. The defect was more common on the left side, and the reason is unclear. In addition, the defect was longitudinal, which may have a particular relationship with the direction of the dural fibers.

Although selective closure of the dural defect is effective during operation, detecting it before laminectomy is still challenging [2, 18]. Cinematic magnetic resonance imaging (cine-MRI) can visualize fluid and tissue movement and is a dynamic water imaging technique used to identify a pulsating flow void to detect the dural defect of SEACs [30]. However, Funao et al. reported that dural defects were revealed in only 2 of 12 cases with SEACs by cine-MRI [7]. CT myelography can demonstrate the defect between the cyst and arachnoid space, whereas it is more sensitive in confirming the defect exists than locating it. The time for the contrast agent to pass through the defect is not easy to capture in SEACs, because of CSF entering the cyst intermittently; the flow rate of CSF and the size of

defect vary between persons. In addition, CT myelography is an invasive operation. Therefore, it is not commonly used [4,17,18,31]. Other defect location methods include time-spatial labeling inversion pulse MRI [32,33], magnetic resonance myelography [34,35] and digital subtraction cystography [31]. However, these methods are limited in clinical application due to the frequent negative results.

If preoperative imaging studies fail to identify the defect, intraoperative ultrasound may help to demonstrate the defect of SEACs during surgery [36]. In addition, an ultra-fine flexible endoscope or an angled endoscope can be inserted into the cyst for observation during surgery to identify the defect location [37,38]. Intraoperative MR myelography has also been used for detecting dural defect [34]. During operation, the position of the dural defect can be determined by the Valsalva maneuver, and it can also be used to evaluate the water tightness of the dural defect suture [18,39,40]. The problem is that these techniques are only helpful intraoperatively, often requiring a laminotomy for exposing the cyst.

We reviewed 32 articles, including 145 cases, and found that the defect position of SEACs is usually at the middle level or above the middle level of the cyst (Supplemental Table S1), and the results are shown in Fig. 6. These cases confirm the feasibility of our method of locating the dural defect through the middle level of the cyst. This method does not rely on special imaging examinations, such as cine-MRI, CT myelography, and intraoperative ultrasound. A possible explanation for this might be as follows. According to Pascal's law, $P = \rho gh$, the pressure of the same liquid in all directions at the same depth is equal. Therefore, as shown in Fig. 7, comparing the subarachnoid space of the brain and spinal cord to a closed container, the hydrostatic pressure generated by CSF at point P, which represents the dural defect, is equal in the upper pole (point A) direction and the inferior pole (point B) direction because of the same height difference H. This means that h1 equals h2 when the cyst initially forms, but as the cyst progresses, the pressure at point B will be higher than that at point A because of the height difference h', so h2 will gradually be larger than h1. However, since h' is much smaller than H, h2 will not be much larger than h1. The above defect location method is unsuitable for all cases, especially multi-cysts, due to the interaction force between the cysts.

Small pits formed in the inner plate of the lamina due to the continuous CSF impingement at the defect site are different from the thinning of the lamina of the non-defect spinal segment (Fig. 5E and F). Preoperative three-dimensional CT can detect this change, which also helps to locate the defect.

There were no cases of non-communicating SEACs in our series, which was rarely reported in the literature [24,39,41,42]. The non-communicating SEACs likely develop directly from communicating SEACs, which may result from the closure of the dural defects by the proliferation of arachnoid cells [24]. According to the Laplace law, Kim et al.41 believed that the dural defect would likely have closed as the cysts continued to expand. When the cyst's pressure increases higher than in the arachnoid space, the CSF cannot open the one-way valve. As time goes on, the dural defect gradually closes [42].

4.4. Limitation

Our method of locating dural defects is not suitable for multiple cysts. It also cannot determine which side of the dural defect is

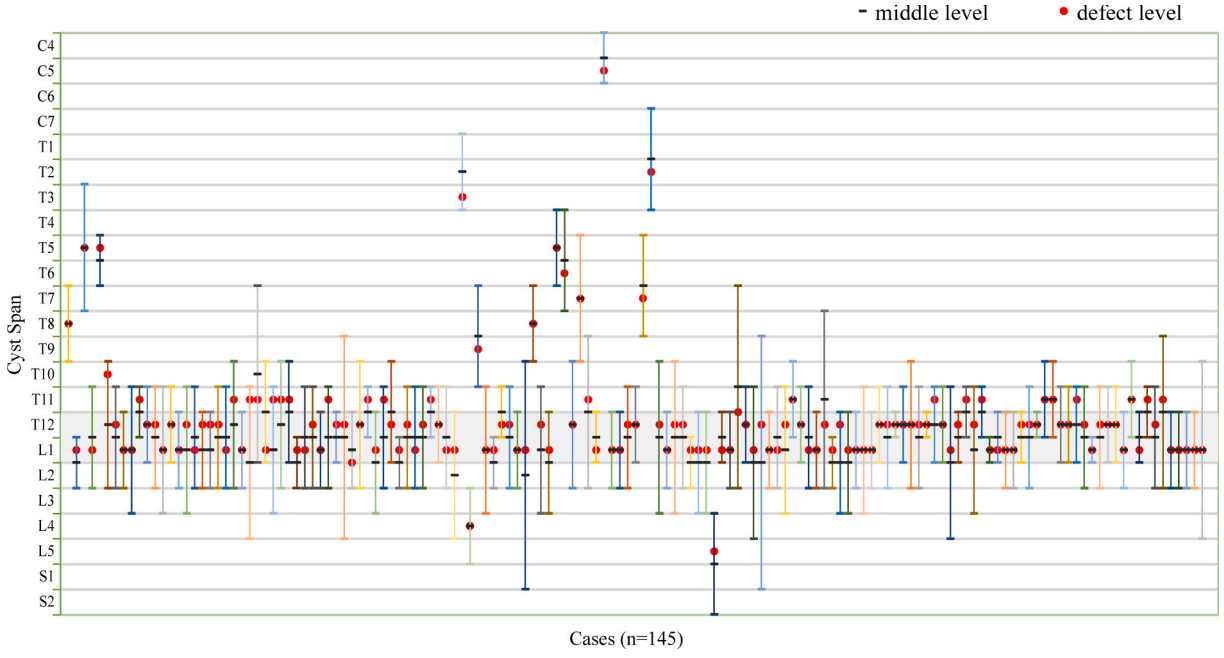

Fig. 6. Cyst span and dural defect location of SEACs reported in the literature. We reviewed 32 articles, including 136 cases, and found that the defect position of SEACs is usually at the middle level or above the middle level of the cyst.

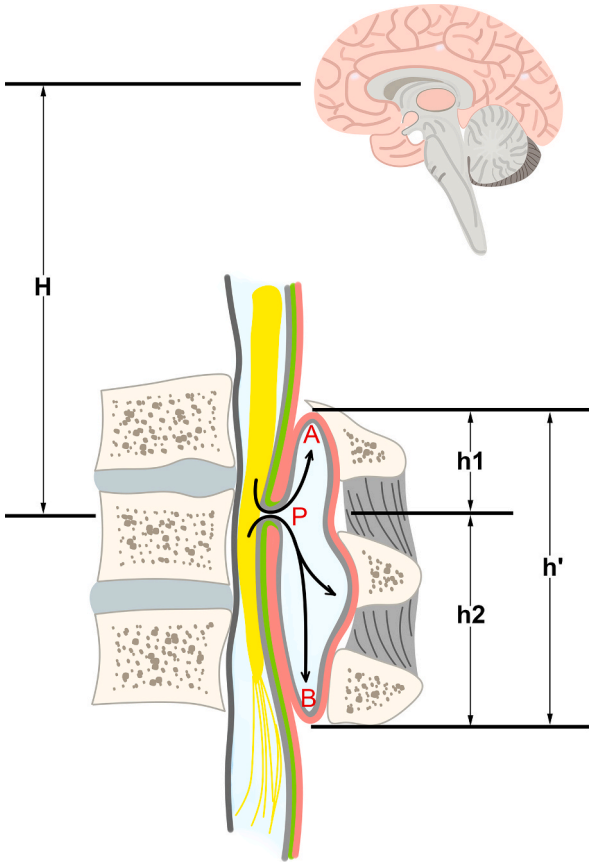

Fig. 7. A possible explanation for the defect position is usually at the middle level or above the middle level of the cyst in SEACs.

located in the spinal canal. Due to the small number of cases included in this study, we reviewed 32 articles, including 145 cases, to confirm the feasibility of our localization method. Since no specific scores, such as Oswestry Disability Index (ODI) and SF-36 score, were recorded for all patients, our study was also limited in patients' self-assessment of symptom improvement.

5. Conclusions

SEACs are rare spinal space-occupying lesions that usually occur at the thoracolumbar junction and communicate with the subarachnoid space through a dural defect. Dural defect closure was preferred over total cyst excision because of fewer spinal segment exposure and minor surgical trauma. According to intraoperative and pathological findings, the dural outer layer cyst (DOLC) is a more reasonable hypothesis about SEACs formation. When CT myelography or cinematic MRI cannot detect the location of the dural defect preoperatively, it can be located according to the middle level of the SEACs with high accuracy.

Author contribution statement

He Huang: Conceived and designed the experiments; Analyzed and interpreted the data; Wrote the paper.
Min Wei: Performed the experiments; Analyzed and interpreted the data.
Quanwei Zhou: Contributed reagents, materials, analysis tools or data.
Renjun Peng: Analyzed and interpreted the data.
Xiping Ding: Performed the experiments.
Jian Xi: Conceived and designed the experiments; Wrote the paper.

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Data availability statement

Data included in article/supp. material/referenced in article.

Declaration of interest's statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Not applicable.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.heliyon.2023.e12969.

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