World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(REVIEW ARTICLE)

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# Pediatrics spinal dural diverticulum: A case-based review of nosology, pathophysiology and microsurgical management

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World Journal of Advanced Research and Reviews, 2026, 30(03), 204-208

Publication history: Received on 23 April 2026; revised on 29 May 2026; accepted on 01 June 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.30.3.1565

## Abstract

**Background:** Spinal dural diverticula (SDD), historically identified as spinal extradural arachnoid cysts (SEACs), are rare but significant causes of myelopathy in children. These lesions result from a focal dural defect that facilitates arachnoid herniation and subsequent cerebrospinal fluid (CSF) accumulation.

**Case Description**: An 11-year-old boy presented with a 4-month history of progressive spastic paraparesis (sensory level T4). Thoracolumbar MRI revealed a T4–T7 extradural cystic lesion causing spinal cord compression. Microsurgical exploration via laminoplasty identified a lateral dural defect. Following ligation of the communication and in toto resection, the patient achieved complete neurological recovery.

**Conclusion**: Modern nomenclature favors the term "dural diverticulum" over "arachnoid cyst." Surgical success depends on the meticulous identification and ligation of the communicating ostium to prevent recurrence.

**Keywords:** Child; Myelopathy; Spinal Dural Diverticulum; Duropathy; Microsurgery; Dural defect

## 1. Introduction

Spinal dural diverticula (SDD) represent a rare clinical entity, accounting for approximately 1% of all spinal space-occupying lesions. While they can occur at any age, symptomatic cases in the pediatric population are particularly rare and often present diagnostic challenges. Historically classified by Nabors et al. as Type IA spinal meningeal cysts, the understanding of these lesions has shifted [1-3]. Contemporary neurosurgical paradigms, notably influenced by the work of Klekamp [4], (2017), redefine these entities as focal manifestations of a structural "duropathy." Unlike true cysts, these are diverticula protrusions of the arachnoid membrane through a pre-existing dural defect.

## 2. Case Presentation

An 11-year-old boy presented with a 4-month history of progressive bilateral lower extremity weakness leading to gait clumsiness. Neurological examination revealed spastic paraparesis with a sensory level at T4. Clinical signs included absent abdominal reflexes and bilateral plantar extension (Babinski) responses.

Spinal radiographs demonstrated a widening of the interpedicular distance, suggesting chronic bony remodeling. Thoracolumbar MRI identified a T4–T7 extradural cystic lesion, which was homogeneous, hypointense on T1-weighted images, and hyperintense on T2-weighted images. No contrast enhancement was observed. Conventional MRI sequences failed to visualize the specific site of communication.

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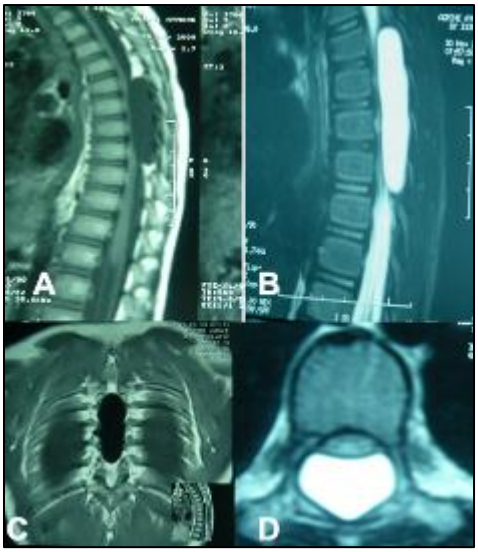


**Figure 1** (A) Sagittal T1-weighted spinal magnetic resonance imaging and (B) T2-weighted magnetic resonance imaging of the thoracic spine showing homogenous cystic lesion at T4-T7 level, caused spinal cord compression. The spinal cord is displaced to the anterior of the spinal canal. (C) Coronal posterior T1- weighted view showing hyposignal cystic lesion. On axial T2-weighted image showing homogeneous and demonstrate high signal intensity (D). Spinal Thoracic Extradural arachnoid cyst in 11-year-old child

A posterior approach was performed via laminoplasty. Under microscopic magnification, a single communicating defect was identified laterally. The communication was securely ligated with a 4.0 Prolene, and the lesion was removed *in toto*. Histological analysis confirmed a thin layer of arachnoid cells. The patient experienced a complete neurological recovery and was discharged 4 days post-surgery.

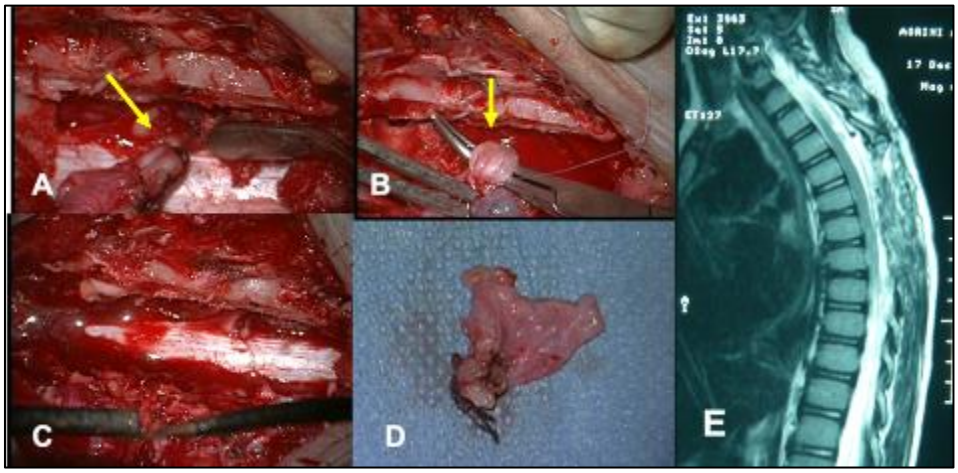


**Figure 2** (A) An intraoperative photograph taken after laminotomy. Intraoperative view on microscope showing communication between cyst and dura (Yellow arrow). A small defect is dissected and ligated (A, B). Final view of the spinal cord with excellent decompression (C) and the cyst (D) with ligature. Postoperative conventional T2-weighted magnetic resonance imaging in the midsagittal plan showing total removal of arachnoidian cyst and good decompression of spinal thoracic cord (E)

## 3. Discussion

### 3.1. Historical Evolution and Nosological Concepts: From "Cyst" to "Duropathy"

The conceptualization of spinal cystic lesions has undergone a profound transformation over the last century, moving from purely descriptive morphological terms to a more precise pathophysiological understanding.In the early 20th century, these lesions were often described as "extradural arachnoid cysts" (EAC) or "meningeal diverticula." The focus

was primarily on the presence of a fluid-filled sac compressing the neural structures. Early authors viewed these as independent pathological growths, similar to intracranial arachnoid cysts, where the cyst wall was considered the primary anomaly.

A major milestone occurred when Nabors et al. (1988) introduced a comprehensive classification that standardized the terminology for spinal meningeal cysts. They categorized them into three types:

- **Type I:** Extradural cysts without nerve root fibers (Type IA: Extradural arachnoid cysts; Type IB: Sacral meningocele).
- **Type II:** Extradural cysts containing nerve root fibers (Tarlov cysts).
- **Type III:** Intradural arachnoid cysts. While this classification was globally adopted, it remained predominantly anatomical, focusing on the location and contents of the cyst rather than its mechanical origin.

The most recent paradigm shift, spearheaded by **Klekamp (2017)**, argues that the term "cyst" is a misnomer for these lesions. Unlike true cysts, which are closed epithelial-lined cavities, these lesions are protrusions or **diverticula** of the existing arachnoid and dural planes. Klekamp introduced the concept of **"Spinal Duropathy,"** suggesting that the primary pathology is a structural failure or focal weakness of the spinal dura mater. This defect, whether congenital (linked to mesenchymal developmental anomalies) or acquired (post-traumatic or iatrogenic), allows the arachnoid membrane to prolapse into the extradural space.

This evolution in nomenclature is clinically significant: it redefines the surgical objective. If the lesion is a "cyst," the goal is resection; however, if it is a "diverticulum" or a "duropathy," the priority is **dural repair** (the closure of the ostium). This conceptual shift has paved the way for modern, minimally invasive techniques focused solely on dural ligation.

### 3.2. Pathophysiology: The Check-Valve Mechanism and Fluid Dynamics

The expansion of SDD is driven by a complex interplay of hydrostatic and hydrodynamic forces, described as **The Ball-Valve Effect:** CSF enters the diverticulum during peaks in intrathecal pressure (Valsalva maneuvers, coughing, or arterial pulsations). Due to the narrow and often oblique nature of the dural neck, the arachnoid membrane acts as a one-way valve, trapping fluid during pressure troughs. Furthermore, high-resolution flow studies indicate that systolic pulsations are amplified within the diverticulum, creating a chronic "hammer effect" on the spinal cord.

### 3.3. Advanced Diagnostic Imaging

On standard T1-weighted and T2-weighted sequences, SDDs typically appear as extradural collections that are isointense to cerebrospinal fluid (CSF). Key morphological signs include spinal cord compression, displacement of the posterior dura mater (the "dura sign"), and widening of the interpedicular distance due to chronic pressure. However, identifying the precise site of dural communication remains the primary diagnostic hurdle, with conventional MRI failing to localize the ostium in more than 70% of reported cases.

To overcome these limitations, advanced flow-sensitive sequences have become essential: Cine-MRI (Phase-Contrast MRI): By synchronizing image acquisition with the cardiac cycle, Cine-MRI provides a dynamic view of pulsatile CSF flow. The detection of turbulent flow at the dural interface is a strong indicator of the defect's location, allowing for a more targeted surgical approach [4].

Intraoperative Adjuncts: When imaging is silent, intraoperative Doppler ultrasonography can detect pulsatile flow through the dural wall, guiding the surgeon directly to the ostium [5].

### 3.4. Meta-analysis of Surgical Strategies and Global Outcomes

The surgical management of spinal dural diverticula (SDD) has undergone a major transition, moving from aggressive resections to targeted micro-anatomical repairs. This section synthesizes contemporary paradigms by evaluating their efficacy, safety profiles, and functional outcomes.

#### *3.4.1. Comparative Analysis of Operative Paradigms*

- **Radical Resection vs. Selective Ligation (The "Neck-Only" Approach)** Historically, the standard of care—supported by series such as Garg et al. (2025) [6] and Woo et al. (2016) [7]—consisted of the complete excision of the diverticulum wall. The rationale was twofold: ensuring immediate spinal cord decompression and obtaining a complete specimen for histopathological confirmation. However, this approach often requires

extensive dissection of the epidural venous plexus, increasing blood loss and operative time. Conversely, a paradigm shift led by Lee et al. (2012) [8] and Neo et al. (2004) [4] suggests that total resection is not imperative. Their "selective ligation" technique focuses exclusively on the dural ostium. Once the communicating "neck" is ligated, the diverticulum collapses spontaneously due to the cessation of the CSF pressure gradient. Comparative series indicate that selective ligation yields equivalent neurological recovery rates with significantly lower surgical morbidity.

- **B. Laminoplasty vs. Laminectomy in the Pediatric Population** A critical factor in pediatric neurosurgery is the maintenance of sagittal alignment. Tsuchimochi et al. (2019) [10] demonstrated that multi-level laminectomies are associated with a 35% to 40% risk of postoperative kyphosis or spinal instability in children. Consequently, osteoplastic laminoplasty has become the gold standard. By repositioning and fixing the posterior elements, surgeons prevent the formation of the "post-laminectomy membrane" and preserve the biomechanical integrity of the growing spine [3-5,9].

*3.4.2. Technical Innovations and Adjuvant Tools*

- **Contribution of the Operating Microscope and Endoscopy** Identifying the dural defect remains the critical point of the procedure. The use of high-magnification microscopy is now indispensable. Liu et al. (2007) [9] highlighted that the defect is often located laterally or near the nerve root emergence, making it invisible to the naked eye. Furthermore, Ouyang et al. (2022) [11] introduced the auxiliary neuroendoscopic approach. This allows for exploration of the extradural space via a single-level laminectomy, enabling the localization and ligation of defects spanning several vertebral segments without expanding the bone opening.
- **Intraoperative Localization Techniques** When preoperative imaging (Cine-MRI or T-SLIP) is inconclusive, intraoperative adjuvants become vital. Kanetaka et al. (2011) [5] successfully used micro-Doppler ultrasonography to detect CSF jets through the dural wall. Other teams have reported the use of intraoperative Valsalva maneuvers or intrathecal fluorescein injection to visualize the leak in real-time under the microscope.

*3.4.3. Synthesis of Results and Prognostic Factors*

A meta-synthesis of clinical results from major institutional series reveals the following data

- **Neurological Recovery:** More than 92% of pediatric patients achieve complete recovery or significant improvement (McCormick score or Nurick grade) within 6 months following surgery. The pediatric spinal cord demonstrates remarkable plasticity once the hydrostatic pressure is relieved.
- **Recurrence Rate:** Recurrence is rare (< 3%) and is almost exclusively attributed to overlooked secondary communications. Approximately 5% to 10% of SDD cases are multi-segmental with multiple dural orifices (Type IB).
- **Complications:** The most frequent complications include transient pseudomeningocele (2–4%) and superficial infections. Long-term spinal deformities are significantly reduced by the systematic adoption of laminoplasty.

## 4. Conclusion

Pediatric spinal dural diverticula are reversible causes of myelopathy. The shift toward recognizing these as structural duropathies emphasizes dural repair as the primary goal. Microsurgical ligation of the communicating ostium, ideally via laminoplasty, ensures excellent functional outcomes and prevents recurrence.

## Compliance with ethical standards

### *Disclosure of conflict of interest*

No conflict of interest to be disclosed.

### *Statement of informed consent*

Informed consent was obtained from the family of child.

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