Spinal arachnoid cysts (SACs) are rare entities which can arise throughout the spinal column in children or adults. Most commonly these lesions are extra-dural, however intra-dural and even intra-medullary lesions are well described in the pediatric literature which consists mainly of case reports or case series [1–23]. Most often, SACs are found after months or years of symptoms such as progressive lower extremity weakness, back pain, gait spasticity, or other signs of myelopathy, and the management strategy of choice is surgical excision or fenestration of the lesion with restoration of normal cerebrospinal fluid (CSF) flow. Due to the increasing use of routine magnetic resonance imaging (MRI) within the pediatric population, these lesions are at times now found incidentally in an asymptomatic patient; the management strategy and natural history of these lesions are not well-established. The acute onset of symptoms from SACs is exceedingly rare. Here, we report a case of acute paraplegia following mild trauma in a young girl with a dorsal thoracic intradural SAC.

1. Case report

1.1. History and examination

An 18-month-old girl was brought to our hospital's emergency department with acute onset paraplegia. She was otherwise healthy and had developed normally prior to hospitalization. She first walked at approximately 1 year of age and had no gait difficulty prior to presentation. There was no family history of neurological illness or arachnoid cyst.

The evening prior to admission, the child was jumping onto a short plastic children's chair when the seat broke, causing her to fall approximately one foot. She landed first on her feet and immediately fell into a seated position. After the mild trauma, her mother noted some gait clumsiness but the patient was ambulatory. She did not complain of severe back pain. Shortly thereafter she was put to bed.

The next morning, the child awoke unable to move her legs. The family brought her immediately to a local hospital and she was then emergently transported to our facility. We observed flaccid paralysis of the lower extremities and bilateral Babinski signs in our hospital's emergency department. Knee and ankle reflexes were appreciated. The patient was ambulatory. She did not complain of severe back pain. Shortly thereafter she was put to bed.

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After several months of physical therapy the child achieved meaningful neurological recovery. Spinal arachnoid cysts can cause acute decompensation in children with serious neurological injury following mild trauma, this risk should be weighed when managing asymptomatic lesions.
Pre-operatively, the cord itself had significant swelling and T2 signal change on MRI. She was taken emergently to the operating room for decompression of the spinal cord in an attempt to maximize functional recovery.

1.2. Operation

Electrophysiological monitoring was established, and initial baseline motor evoked potentials and somatosensory evoked potentials (SSEPs) were absent in the lower extremities prior to skin incision. A standard thoracic laminoplasty was performed, with exposure of T3-T9. The operative microscope was brought into use, and the dura was carefully opened. In the caudal direction of exposure was a thickened arachnoid membrane with clear CSF. There was no evidence of acute or subacute hemorrhage. The venous system of the dorsal spinal cord appeared full and the fluid here was not under significant pressure. However, in the rostral aspect of our exposure, a thinner arachnoid membrane was visualized as well. This was entered sharply, and CSF was expressed under significant pressure. At this rostral site, the dorsal spinal cord vasculature appeared blanched and the cord itself was displaced ventrally (Fig. 2). Upon decompression at this rostral aspect of the lesion, there was a slight improvement in SSEPs, but no return of motor potentials. After resection of the visualized arachnoid membranes and ensuring good CSF flow in all directions, the dura was closed, the lamina were replaced using suture, and the wound was closed in a standard multilayer fashion.

1.3. Postoperative course

Post-operatively, the child returned to the intensive care unit where Mean Arterial Pressure (MAP) was kept elevated (>85 mm Hg) for five days to maximize spinal cord perfusion. An MRI was performed demonstrating excellent decompression (Fig. 3) but with significant progression in spinal cord signal change and edema, consistent with infarct. After one month of intensive therapy, the patient had regained 50% of motor function and the patient was discharged home with a rehabilitation plan.

Fig. 1. Axial (A, B) and sagittal (C, D) preoperative T2-weighted MR images demonstrate a dorsal fluid collection consistent with arachnoid cyst (White arrow) in the thoracic spine compressing the spinal cord with early spinal cord (Black/gray arrow) signal change.

Fig. 2. Intraoperative microscopic view demonstrating operative findings including paucity of dorsal vessel filling with blanched cord (Black arrow) at site of high-pressure CSF space, remnant of thickened arachnoid membrane caudally (Gray arrow), and caudal cord under the arachnoid cyst with normal blood vessel filling and cord color (White arrow).
inpatient acute rehabilitation the child was discharged with no return of lower extremity function but with some response to painful stimulus. Subsequently, the patient continued with 2-3x of weekly outpatient physical therapy. After an initial 8-week follow-up, the child regained some spontaneous non-purposeful movement of the lower extremities but no functional strength and remained paraplegic. We continued to monitor the progress of the patient’s recovery who has since completed 10 months of 2-3x of weekly outpatient physical therapy. To our surprise, at the most recent follow-up (12 months post injury), the child now has voluntary movements in the bilateral lower extremities including hip flexion. Although the patient continues to be paretic, the patient is also able to stand with assistance, bear weight, and is able to walk short distances with the aid of a walker. Additionally, the child has normal urodynamic testing and exhibits excessive toe biting, suggestive of some sensory function and may be due to dysesthetic pain and paresthesia.

2. Discussion

Spinal arachnoid cysts are uncommon lesions in children that often present with progressive signs and symptoms of myelopathy due to cord compression. Acute symptoms from a previously asymptomatic SAC are exceedingly rare; we found three cases in the literature where a child clearly presented with acute onset (<48 h) of weakness after a mild traumatic injury. One publication reported two cases of patients with ventral cervical SACs presenting with tetraparesis or tetraplegia after mild trauma [17]. Another case series of three patients with SACs described a single pediatric patient with acute onset tetraplegia after mild trauma with a cervico-thoracic junction ventral SAC [15]. We found no case reports of dorsal SACs causing acute symptoms after trauma. Overall, the majority of published literature that we reviewed consisted of patients with subacute or chronic symptoms (Table 1).

These lesions are generally considered slow growing, and the etiology of acute onset deficits in children is not obvious. One possibility would be a direct cord contusion from the traumatic insult, in the setting of acquired spinal stenosis from the SAC. This

**Table 1**

Review of pediatric spinal arachnoid cyst published series.

<table>
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<th>Author</th>
<th>Year</th>
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would be akin to a spinal cord injury mechanism similar to that of adult patients with thoracic herniated disc disease who present with acute spinal cord injury after a mild trauma. However, we speculate that a blunt cord injury such as this would be unlikely to occur over multiple levels such as in our patient. Based on the intraoperative findings of a CSF collection under significant pressure, with obvious ongoing cord compression, we suspect a ball-valve mechanism whereby the mild traumatic injury caused a tear within the trabecule of the arachnoid cyst. Unidirectional flow of CSF into such a space then would create an ongoing, expansive mass lesion causing cord compression and ischemia. We believe this theory is more fitting with the intraoperative findings of thickened arachnoid (i.e., the chronic, lower-pressure SAC) below the level of cord compression, and CSF under high pressure within a separate cystic space at the level of cord compression. This theory has also been postulated for the initial formation of SACs, and an exaggerated but similar mechanism via a traumatic tear in the arachnoid could explain acute decompensation [1, 3].

As more and more pediatric patients undergo MRI, the incidence of asymptomatic SACs will undoubtedly increase. The optimal management of these asymptomatic lesions is not clear. Some have recommended yearly monitoring with imagining to evaluate for new signal changes and confirm the benign nature of the lesion [24]. However, no consensus exists on the natural history of asymptomatic SACs in the pediatric population, and some may regress or remain stable over years without symptoms.

When SACs present with progressive, chronic symptoms, the choice for surgical decompression to maximize recovery and prevent progression is clear. We found only one case report in which the treating physician opted for conservative management even in the case of mild symptoms [25]. The patient was in his early teens when intermittent symptoms of pain and paresthesiae began, however they were not progressive and underwent no intervention. The patient re-presented as an adult with mild progressive symptoms of pain over the left thigh over the course of one year. An MRI of the patient’s spine demonstrated a posterior extradural cysts at the thoracolumbar junction; however due to the mild symptoms the patient and physician did not pursue any surgical intervention and no specific medical treatment was discussed [25]. This was a rare entity and was not associated with trauma; in instances in which there is clear acute progression of neurological injury we believe surgical management remains the responsible choice. In two of the largest series of pediatric patients who were managed with either surgical excision or fenestration, 87%–94% had symptomatic improvement or complete remission [1, 6]. Open microsurgical fenestration is a more commonly reported procedure over complete excision; however recurrence rates for this procedure ranged from 3 to 12.5% [1, 6]. In cases of recurrence, cystoperitoneal shunting has been successfully used as a last resort treatment [1, 6, 13, 26]. Recently, minimally invasive endoscopic and MRI guided needle fenestration procedures have been reported [4, 5, 23]. These procedures have similar outcomes when compared to open surgical intervention, however they have the added benefit of limited blood loss, reduced operative time, and smaller incision and subsequent scar formation [5].

The presented case illustrates the potential for meaningful recovery after acute decompensation and para- or tetraplegia after mild trauma. We were pleasantly surprised by the neurological recovery that was made by our patient after completing several months (1 month inpatient, 7 months outpatient) of aggressive neurorehabilitative therapy. This recovery potential may be unique to the pediatric population. In a large series of severe pediatric spinal cord injuries, functional recovery occurred at higher rates in children (64% vs 31.4%) when compared to adults with similar injury severity at 1 year follow-up [27]. Additionally, functional gains in pediatric patients are dependent on the severity of the initial injury, thus, limiting the acute progression of a neurologic injury is vital for improving outcomes [28]. Following surgical decompression, our patient’s MAP was kept elevated (>85 mm Hg) for 5 days to ensure adequate profusion of the spinal cord. Maintaining and elevated MAP after spinal cord injury (SCI) has been shown to improve functional outcomes in patients with SCI, which may be due to limiting the extent of secondary injury caused by ischemia or local profusion changes [29–31].

3. Conclusion

As indicated by our literature review, the onset of acute symptoms following mild spinal trauma in patients with previously asymptomatic spinal arachnoid cyst is exceedingly rare. Although there is clear benefit from surgical intervention in symptomatic patients, the management of asymptomatic patients remains ill-defined. This case however illustrates the potential risk of acute symptomatic progression in this patient population, and makes a compelling case for early and a more aggressive surgical approach to prevent future neurological catastrophe in asymptomatic patients.

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References


