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Secondary tethered cord syndrome in patients with myelomeningocele

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Abstract. Myelomeningocele is associated with other neurological abnormalities, including hydrocephalus, Chiari II malformations, syringomyelia, and secondary tethered cord syndrome. Tethered cord syndrome occurs because of abnormal attachment of the spinal cord to the caudal dural sac, causing cord ischemia. Occasionally, symptoms of progressive neurological deterioration may occur and can significantly affect the long-term outcome of these patients. Proper management of patients with myelomeningocele requires long-term follow-up and evaluation of signs of neurological deterioration that suggest secondary tethered cord syndrome. Treatment of these patients may target symptoms, such as urological intervention for bladder dysfunction, or it may target the tethered cord itself. Recently, many studies have shown that tethered cord release can significantly improve symptoms in these patients.

Keywords: Myelomeningocele, tethered spinal cord, retethering, tethered cord release, pediatric neurosurgery

1. Introduction

Myelomeningocele (MM) is an uncommon congenital malformation (incidence of 0.41–1.43 per 1000 live births) requiring life-long medical care [5]. MM is associated with numerous neurological abnormalities including hydrocephalus, Chiari II malformations, syringomyelia, and tethered cord syndrome. Proper management of these associated conditions may significantly improve outcomes in this population. In developed nations, due to early primary surgical repair and close follow-up by a multi-disciplinary team of physicians, a large percentage of patients with MM can be expected to reach adulthood, with 82% of survivors achieving independence in their activities of daily living [5]. Therefore, the appropriate management of MM-associated abnormalities is essential in order to decrease morbidity and maintain functional independence for these patients.

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Tethered cord syndrome (TCS) is a progressive, neuro-degenerative disorder of the spinal cord. It is a common consequence of several dysraphic lesions of the spine, including myelomeningocele. During normal movements such as flexion and extension, the length of the spinal canal changes. As a result of this, the spinal cord must normally move with respect to the spinal canal. The spinal cord is attached caudally to the filum terminale, a thin band of connective tissue that extends from the caudal end of the spinal cord to the inferior termination of the spinal canal. This attachment of the spinal cord to the filum terminale does not normally apply significant traction to the cord itself during normal movements. Similarly, although nerve roots leave the cord at each functional spinal level, these do not normally tether the cord in an anatomic position. Normally, therefore, the spinal cord is not physiologically tethered by its attachments within the caudal dural sac. In contrast, in cases of TCS there is an abnormal attachment of the cord, most often caudally, to surrounding structures. These abnormal attachments limit movement of the cord [8,16,27]. In animal models, this tethering and subsequent traction of the spinal cord during motion of the spinal canal has been shown to cause derangements in blood flow

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Table 1
NEM Neurological Scale used for follow-up [20]

Score	Motor	Sensory	Bladder	Anus
1	Wheelchair	Skin ulceration or amputation	Incontinence day and night	Incontinence
2	Major orthesis or two crutches	Pain	Noctural Incontinence	Painful constipation
3	Minor or distal orthesis	Painless sensory deficit	Intermittent Catheterization	Normal
4	Fatigue on Walking	Normal	Dysuria, infections, or stress incontinence	–
5	Normal	–	Normal	–

and oxidative metabolism, ultimately leading to cord ischemia and worsening neurological function [28,32]. Furthermore, spinal cord blood flow has been shown to increase following tethered cord release [26].

TCS often presents during periods of growth, most frequently between the ages of 6–13 years [8,15]. It is thought that worsening injury to the tethered spinal cord occurs as the spinal canal lengthens during growth. Occasionally, however, symptoms do not correlate with growth spurts, and patients may even present in adulthood with new symptoms of tethered cord [13,29].

After initial repair of a MM, most patients will have spinal cords that are anatomically and radiographically tethered. Significantly, only a minority of patients will become symptomatic. It is this group of patients with both anatomic evidence of tethering as well as concordant symptoms that suffer from what is called tethered cord syndrome. The term TCS is typically not applied to the larger group of patients with only radiographic evidence of tethering in the absence of symptoms.

Tethering occurs in MM patients because of the presence of surgical scar on both the spinal cord as well as its coverings, as well as the distorted anatomy of the spine, the nerve roots, and the dura. Therefore, most commonly, a cord tethers at the site of previous MM repair [8]. Arachnoiditis and tight dural closures may increase the risk of recurrent tethering [16]. Occasionally, dermoid cysts may arise at the site of prior repair and can contribute to the onset of symptoms. These cysts arise from dermal rests that are trapped within the closure during primary repair of the MM.

Although this review focuses on TCS that develops after primary repair of MM, it is important to emphasize that TCS can occur in the setting of other spinal pathologies, all of which have a similar presentation. Secondary tethered cord syndrome (STCS) is defined as symptoms of tethered cord presenting in a delayed manner following initial untethering or initial repair of a primary spinal lesion. Examples of primary lesions include intradural lipomas, diastematomyelia, dermal sinus tracts, tight filum, dermoid tumors, sacral agenesis, and myelomeningoceles [8,16].

Given that STCS is an often slow, progressively deteriorating process which is found in a population whose neurologic function may change over time, the true prevalence of STCS is difficult to determine. The prevalence of STCS in MM has been reported as low as 3% or as high as 32% [1,16,19,29]. After surgical untethering of STCS, many patients again anatomically and radiographically tether, however most of these remain asymptomatic. Unfortunately, some patients may experience new symptoms or deteriorating function from repeat tethering even following a previous STCS repair. These patients present with many of the same symptoms and clinical findings of an initial tethered cord. The prevalence of recurrent symptomatic tethering, sometimes called multiple repeat tethered cord syndrome, following an earlier STCS repair is approximately 20% [15,27].

2. Diagnosis and evaluation

2.1. Diagnosis

At many centers, patients with MM are evaluated on a yearly basis by a multidisciplinary team of surgeons, neurologists, and physiatrists. A complete history and physical examination, with the use of any relevant studies are carried out in order to detect any evidence of neurological deterioration, including that caused by STCS. Often subtle changes on history and examination are difficult to appreciate, and thus the consistent use of objective scales can aid in early diagnosis of STCS (Tables 1–4) [9,14,20,27]. Many of these measures are straightforward and can even be routinely completed by a patient's family. Currently, there are no data identifying the most clinically effective and sensitive scales.

Although signs and symptoms of STCS are well recognized by physicians, strict diagnostic criteria do not currently exist [6,29]. The characteristic features of STCS are a progression of symptoms and a deterioration of neurologic status (Table 5). The most common presentation is back pain or deterioration of motor function [16], but patients can present with many combinations of signs and symptoms. New or progressive

Table 2
NEM Neonate and Infant Neurological Scale [20]

Score	Motor	Sensory	Bladder	Anus
1	Major Deficit	–	Incontinence	Incontinence
2	–	Pain	Retention	–
3	Club Foot, Atrophy, or Distal Deficit	–	–	Normal
4	–	Normal	Dysuria, infections, or stress incontinence	–
5	Normal	–	Normal	–

Table 3
Urinary Leakage Scale used for follow-up [14]

Score	Symptom
1	Permanent Leak
2	Frequent Leak
3	Occasional Leak
4	Rare Leak
5	Never Leak

symptoms or signs suggestive of STCS should instigate a thorough work-up (Table 5). When evaluating a patient's history, symptoms of pain, bladder and bowel dysfunction, sensory loss, and changes in ambulation or gait should be specifically assessed [1,6,8,29,30]. Back or leg pain may indicate significant underlying pathology and requires a careful assessment in this population. In fact, although pain symptoms are subjective and notoriously difficult to quantify, pain alone can indicate a symptomatic tethered cord. Patients with STCS often present with worsening incontinence, delayed toilet training, or recurrent febrile urinary tract infections. Occasionally, symptoms of progressive constipation or encopresis can be seen as well.

A proper physical examination includes full upper and lower extremity motor examination, sensory and skin examination to evaluate for loss of sensation or skin breakdown, and examination of the spine and lower extremities to evaluate for evidence of scoliosis, back tenderness, or progressive lower extremity deformities. A change in gait, posture, or any new difficulty with ambulation can be a sign of neurologic motor deterioration, spasticity, or an orthopedic deformity of the lower extremity. New or progressive painless sensory loss, dysesthesia, or recurrent decubitus ulcers can also suggest STCS [20]. Evidence of kyphotic or scoliotic deformities on exam should prompt periodic radiographic evaluation, with progression of the deformity initiating an evaluation for other signs of STCS.

In the evaluation of these patients, emphasis should be placed on assessing urological function, as renal failure has been identified as a significant cause of morbidity and mortality in patients with MM. Twenty percent of the mortality of patients with myelomeningocele in the first year of life is secondary to renal failure [3]. In a long-term follow-up study, Hunt et al. noted that half of their patient population had died by age 35, and 29% of these deaths were due to renal causes [10]. Tarcan et al. have also emphasized the importance of close long-term urological follow-up in the MM population [30]. These authors followed a group of patients with myelodysplasia and normal urological studies initially after birth, and found that with close follow-up and yearly urodynamic studies 32% of their patients were diagnosed with urological deterioration by 6 years of age. Close urological follow-up with urodynamic studies, renal ultrasounds, and urinalyses performed on a regular basis will help preemptively identify worsening bladder function. Urological studies indicating worsening upper urinary tract dilatation, vesicoureteral reflux, decreased bladder capacity, increased bladder hyperreflexia, or decreased detrusor leak point pressures may suggest a diagnosis of STCS [29,30].

2.2. Differential diagnosis

Many of the associated abnormalities seen in patients with MM such as hydrocephalus, Chiari II malformation, syringomyelia, and pseudomeningocele can cause neurological deterioration with symptoms that are similar to those of STCS [5]. Most patients with MM have a ventriculo-peritoneal shunt for hydrocephalus, and shunt malfunction is a frequent cause of neurological deterioration in these patients. When shunts fail, patients can present with a spectrum of clinical manifestations including, most frequently, headaches, vomiting, blurry vision, and irritability. However, these patients can occasionally present with isolated lower extremity symptoms. Shunt malfunction, therefore, may be difficult to diagnose based on history alone. Associated Chiari II malformations may present with symptoms of brain stem dysfunction, including blurry vision or diplopia, depressed gag reflex, and apnea. Patients can also present with signs and symptoms of cord myelopathy, which can include upper and lower extremity manifestations. Syringomyelia and syringobulbia can also occur in this population, causing a range of symptoms depending on its size and location. Pseudomeningocele

Table 4
Modified Hoffer Ambulatory Scale [9,27]

Score	Symptom
1	Non-ambulant
2	Exercise ambulant (only in therapeutic situations)
3	Household ambulant (using crutch or brace, indoor; wheelchair outdoors)
4	Community ambulant (ambulant outdoor with or without brace; uses a wheelchair for longer distances)
5	Normal ambulant

Table 5
Presenting signs/symptoms of STCS with incidences reported from several studies

Presenting Sign/Symptom	Herman et al. [8]	Bowman et al. [1]	Haberl et al. [6]	Maher et al. [15]
Back or Leg Pain	32%	22%	17%	70%
Motor/Ambulatory Dysfunction	100%	39%	*	80%
Urologic Dysfunction	6%	13%	10%	40%
Bowel Dysfunction	*	*	1.3%	*
Sensory Loss	*	*	*	30%
Spasticity	*	26%	68%	*
Kyphoscoliotic Deformity	51%	43%	17%	*
Orthopedic Lower Extremity Deformities	11%	*	*	*

$*$ = Not specifically reported as presenting sign/symptom.

at the site of previous repair must also be included in the differential diagnosis for severe progressive back pain in this population. When patients present with signs or symptoms suggestive of STCS, it may be difficult to identify the cause of deterioration solely based on history and physical examination. Imaging studies can be helpful. Furthermore, a shunt tap can be performed under local anesthesia, or intra-operatively under general anesthesia to evaluate for proper shunt function. At our institution, we routinely perform shunt taps prior to a surgical untethering. In more suspicious cases, a more involved work up including monitoring of intracranial pressure or surgical shunt exploration may be needed.

2.3. Radiologic evaluation

On MRI, signs of a tethered cord include adherence of the cord or cauda equina to surrounding structures and a spinal cord that terminates at a lower than normal level. However, these anatomic features are nearly universal following initial repair of MM. Therefore, MRI findings alone may not be used to define patients with STCS. Nevertheless, MRI may be used to define the anatomy prior to potential surgery, identify the site of tethering, and evaluate for other lesions such as dermoid cysts, syringes, and pseudomeningoceles, all of which are known to occur in this population [5,8]. Before the advent of MRI, CT and CT myelogram were the standard radiologic examinations used in the evaluation of STCS. These modalities can still be used today and are especially helpful in patients who have previously undergone spinal fusion with instrumentation.

3. Natural history

Surgical untethering is the usual treatment for symptomatic STCS at many tertiary care centers. Therefore, the incidence and expected progression of neurological dysfunction in these patients without surgical repair is not well established. Despite this paucity of data, however, the natural history of STCS is thought to be progressive. Phuong et al. evaluated 45 MM patients with STCS who were not surgically untethered [19]. The results of their study demonstrates the deterioration that can be expected without surgical untethering. They reported a rate of neurological deterioration by documenting the number of patients that required "end-organ" symptomatic surgical repair such as bladder augmentation and scoliotic fusion for urologic and orthopedic abnormalities. They followed patients for an average of 12 years. They found that 27.5%, 40%, 60%, and 89% at 1, 2, 5, and 12 years respectively required end-organ surgical repair. Furthermore, 45% of patients who underwent release of contractures without neurosurgical untethering required further contracture release.

4. Treatment

When a patient presents with symptoms of STCS, treatment can address the primary pathology, (i.e. the tethered cord), or the symptoms (i.e. urological intervention for urinary incontinence). Early neurosurgical

untethering may improve long-term outcomes, and referral to a neurosurgeon is a fundamental step in the evaluation of a tethered cord. General surgeons, urologists, orthopedists, and physiatrists also play essential roles in the treatment of STCS.

4.1. Bladder symptoms

There are many surgical and non-surgical options for treating neurogenic bladder. The usual goal of treatment is maintenance of social continence and bladder independence, with the critical long-term goal of preventing renal failure secondary to recurrent infections or post-obstructive nephropathy [3,5,29,30]. Clean intermittent catheterization and anti-muscarinic drugs, used to decrease bladder pressure, are generally the first line of treatment. Many surgical options exist for treatment of low bladder capacity, high bladder pressures, urinary tract obstruction, or incontinence including bladder augmentation, cystoplasty, pelvic sling suspension, or stomaplasty.

4.2. Bowel symptoms

Progressive bowel dysfunction in STCS can be treated with oral laxatives in combination with retrograde enemas. If a bowel program is not effective, the use of anterograde colonic enemas via surgical stomas or fecal diversion may be required [3].

4.3. Spasticity and progressive lower extremity deformities

The goal of treatment is the prevention of ambulatory deterioration and development of decubitus ulcers. Braces and orthoses can be useful in treating both spasticity and orthopedic foot deformities. Physical therapy, oral baclofen, intramuscular Botulinum toxin injections, intrathecal phenol injections, and surgical interventions such as Baclofen pump placement, dorsal rhizotomy, and contracture release are additional options available for the treatment of spasticity [22]. Foot deformities can also be treated with complex orthopedic procedures.

4.4. Scoliosis

In patients with MM, the true etiology of scoliosis and the extent to which STCS plays a role in its pathogenesis is unclear. Furthermore, the proper course of neurosurgical treatment is poorly defined [4]. Several groups have evaluated the effectiveness of tethered cord release without fusion as a treatment for progressive scoliosis in patients with myelomeningoceles. Some studies have shown an advantage to untethering patients with smaller curves [17,21]. For instance, McLone et al. found that in patients with initial curves less than 50 degrees, 96% of curves stabilized or improved at 1 year, and 63% remained stable at long-term follow-up [17]. Pierz et al. also found that in long-term follow-up of patients with initial curves less than 30 degrees, scoliosis improved in 20%, stabilized in 50%, and progressed in 30%, with 20% requiring fusion [21]. These reports, however, have shown that in patients with curves greater than 40–50 degrees or in patients with thoracic level MM, tethered cord release without surgical fusion is ineffective at halting curve progression [17,21].

Many centers advocate prophylactic untethering of the spinal cord prior to or concurrently with sciolosis repair. According to a review by Dias, neurological deterioration resulting from surgical correction of scoliosis is quite rare but may occur occasionally [4,12, 18]. Older articles had recommended untethering followed by fusion 3–6 months later [7]. However, since the spinal cord becomes anatomically retethered shortly after surgical untethering, a long delay prior to fusion may eliminate the advantage of untethering. Currently, the preferred treatment at our own institution as well as many others is concurrent untethering and fusion [7, 24].

4.5. Spinal cord untethering

The indications for surgical cord untethering remain controversial. Historically, because of its progressive nature, many neurosurgeons elected to treat this condition conservatively and avoid surgical untethering. Furthermore, it was previously felt that surgical untethering would not affect long-term outcomes despite showing short-term improvements. There is increasing evidence for the effectiveness of surgery to make a positive impact on this disorder. For this reason, early neurosurgical intervention has become common in many institutions [1,6,8,15,17,25,27,29].

A grading system for the evaluation of the degree of untethering has been developed by Kirollos et al. which

suggests that improved neurological outcomes correlate with the degree of untethering. Therefore, complete circumferential untethering is usually attempted [13]. It is important to note, however, that symptomatic improvement may be seen even following technically challenging cases where complete circumferential untethering is impossible [15]. Intraoperatively, monitoring with somatosensory evoked potentials (SSEP), anal sphincter electromyogram (EMG) and anal pressure sensors are often used depending on the surgeon's preference [6,16]. A wide dural patch is often used in an attempt to decrease the rate of retethering [16]. Although many materials have been evaluated as dural grafts, no particular material has been proven to decrease the recurrence rate of TCS compared with any other. Furthermore many graft types, including polydimethylsiloxane (Silastic), and polytetrafluoroethylene (Gore-Tex) have fallen out of favor due to complications such as hemorrhage or cerebrospinal fluid leak [23]. Other materials that have been used for this purpose include allograft dermis, allograft pericardium, and porous polyethylene (Medpor) [2,6,11, 31,33].

5. Surgical prognosis

5.1. Results

The results of surgical untethering for TCS are generally encouraging. Herman et al. reported that in MM patients with STCS, motor complaints improved in 67% following untethering. Furthermore, pain symptoms improved in 91%, gait improved in 72%, urinary control improved in 33%, and patients with scoliosis improved by an average of 7 degrees [8]. They also found that patients who were followed up closely, diagnosed early, and treated early had 82% improvement in symptoms compared to 54% in those who were not closely followed, suggesting that not only surgical treatment, but also early surgical treatment can alter outcome. Tarcan et al. evaluated the effect of untethering on the urological parameters of recurrent urinary tract infections, upper urinary tract dilatation, vesicoureteral reflux, detrusor leak point, and bladder capacity [29]. At six months, they found that sterile cultures developed in 34%, reflux resolved in 47%, and that bladder capacity and detrusor leak point significantly improved. On evaluation at one year, these findings were not significantly different. Haberl et al. also reported clinical improvement following surgical untethering for STCS [6]. At a mean 4.8 year follow-up, 84% of symptoms stabilized, 26% showed improvement, and 16% further deteriorated after untethering. In a study by Bowman et al., 97% stabilized or improved, with a 71% improvement rate, 26% stabilization rate, and 3% deterioration rate [1]. There has been a single report evaluating surgical outcome following multiple repeated tethered cord release [15]. That study included only those patients that had undergone at least a myelomeningocele repair as well as one prior tethered cord release. The results demonstrated that releasing a retethered cord improved pain symptoms in 86% of patients, urinary symptoms in 50%, and weakness in 75% of patients. In addition, they noted that the result of further operations is less successful and more challenging than previous surgeries, and is associated with increased surgical complications.

5.2. Complications

Cerebrospinal fluid (CSF) leak is the most common surgical complication encountered following tethered cord release, with an incidence of 1–10%. Maher et al. noted no CSF leaks in their MM population that underwent recurrent untethering [15]. Additionally, other wound problems such as dehiscence and infections are known to occur occasionally [1,6,8]. At our institution, we routinely close with a myocutaneous flap and have noted a decreased incidence of cerebrospinal fluid leaks, wound infections, and dehiscences. New onset neurological deterioration resulting from is also possible as a result of the untethering procedure.

6. Conclusion

Patients with MM represent a very complex population that requires life-long medical follow-up and care by a multidisciplinary team of physicians and healthcare workers. The natural history of STCS is one of progressive deterioration, and if left untreated, can be potentially devastating to a population that is now living longer and becoming increasingly independent. Avoiding the significant disability associated with STCS requires early diagnosis and prompt intervention. However, in a pediatric population with an evolving neurological status, slow deterioration can be difficult to identify.

Many reports have found that a large percentage of patients stabilize or even improve after untethering, even incomplete surgical untethering. Given the fre-

quently progressive natural history of STCS, even stabilization may occasionally be viewed as an improvment. Furthermore, despite the fact that a small yet significant percentage will symptomatically retether, these patients have positive outcomes with minimal complications after repeat untethering. However, although many studies have shown stabilization and improvement in patients after untethering, these studies lack control groups and are performed at different institutions for various indications. Ideally, a prospective multicenter control trial comparing the outcomes of neurosurgical untethering with symptomatic treatment would be performed to identify the most appropriate treatment regimen for each indication.

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