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SURGICAL NEUROLOGY INTERNATIONAL
SNI: Pediatric Neurosurgery
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Editor: Sandi Lam, M.D. Baylor College of Medicine; Houston, TX, USA

Review Article

Surgical management of lipomyelomeningocele in children: Challenges and considerations

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Received: 20 October 16 Accepted: 08 December 16 Published: 26 April 17

Key Words: Spinal lipoma, lipomyelomeningocele, spinal dysraphism, pediatric

ILLUSTRATIVE CASES

Case 1

A 3-month-old boy was referred to neurosurgery when a subcutaneous bulge in the lower lumbar region was incidentally noted. Initial workup included an ultrasound of the region that was concerning for spinal dysraphism, including sacral agenesis and an associated intraspinal mass. These findings prompted a lumbosacral magnetic resonance image (MRI), which confirmed the diagnosis of lipomyelomeningocele. The patient was clinically asymptomatic, with normal strength in his lower extremities, no evidence of hydrocephalus, and normal bowel and bladder function. Because he was meeting developmental milestones, he was managed observantly with annual clinical exams, which remained normal. At 3 years of age, he underwent urodynamic studies, which were unremarkable, and he was able to successfully toilet train. At around this time, his parents reported transient morning stiffness in the back and lower legs, which would resolve by the afternoon. This prompted a repeat MRI [Figure 1], which demonstrated the development of a syrinx in the lumbar region of the spinal cord. Surgical debulking and untethering of the lipomyelomeningocele was discussed with the parents, along with the associated risks and potential benefits, and, ultimately, the decision was made to continue expectant management. At 5 years of age, he remains clinically asymptomatic and continues to meet developmental milestones.

Case 2

An 11-day-old girl with an uncomplicated birth was noted by her parents to have a lump on her lower back, prompting further workup by her pediatrician. A physical exam revealed a 4–5 cm soft, nontender mass in the lumbar spine with hyperpigmented changes [Figure 2]. An ultrasound was performed, which showed the absence of sacral lamina and dorsal elements with an associated intraspinal mass and bilateral hydronephrosis. She subsequently underwent an MRI showing a spinal dysraphism of the sacrum associated with a large cystic intraspinal mass, concerning for a lipomyelomeningocele versus a terminal lipomyelocystocele. Her motor exam showed normal strength in both lower extremities but poor deep tendon reflexes and a slightly distended abdomen. Further urological workup revealed the presence of grade 4 vesicoureteral reflux for which she was

Access this article online

Quick Response Code:

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.205268

How to cite this article: Wagner KM, Raskin JS, Hansen D, Reddy GD, Jea A, Lam S. Surgical management of lipomyelomeningocele in children: Challenges and considerations. Surg Neurol Int 2017;8:63.
http://surgicalneurologyint.com/Surgical-management-of-lipomyelomeningocele-in-children:-Challenges-and-considerations/

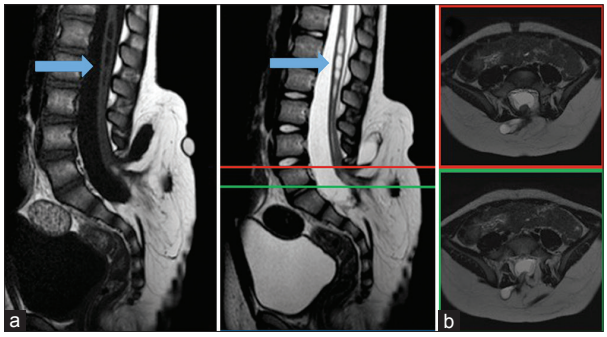

Figure 1: (a) T1 (left) and T2 (right) sagittal sections of an MRI showing the presence of a caudal lipomyelomeningocele with an associated lumbar syrinx (arrows) in a clinically asymptomatic patient. (b) Selected T2 axial sections as identified by the color coding on the sagittal image in A

started on a clean intermittent catheterization protocol. After a thorough discussion about the risks and benefits of operative intervention, she was taken to the operating room (OR) for debulking of the lipomatous mass and untethering. Electrical stimulation and neuromonitoring were used to monitor function. The dural tube was reconstructed using a synthetic patch duroplasty. No cyst was encountered during the debulking. There were no intraoperative complications, however, 1 week postoperatively she developed a cerebrospinal fluid (CSF) leak, which precipitated a *Klebsiella* wound infection. She returned to the OR for irrigation and debridement, placement of a lumbar drain, and revision of the dural closure. Postoperatively, she remained intubated and prone for 1 week, at which point the lumbar drain was removed and she was extubated. She had no further leakage from the wound and was discharged approximately 5 weeks after her first surgery. At 18-months' postoperative follow-up, her wound was well healed, and she retained normal strength in her bilateral lower extremities. She progressed to grade 5 vesicoureteral reflux with moderate hydronephrosis without urinary tract infections, managed by clean intermittent catheterization every 3 hours.

INTRODUCTION

Lipomyelomeningocele (LMMC) is a closed neural tube defect in which neural elements are incorporated into a spinal lipoma. This is an uncommon defect, occurring in 3–6 patients per 100,000 live births.[14] Clinical decision-making regarding treatment is complicated by the varied pathology and the spectrum of presentations. Herein, LMMC embryology, morphology, treatment options, and outcomes are reviewed.

Embryology

Central nervous system development consists of primary and secondary neurulation. During primary neurulation, the notochord induces folding of the neural plate to form

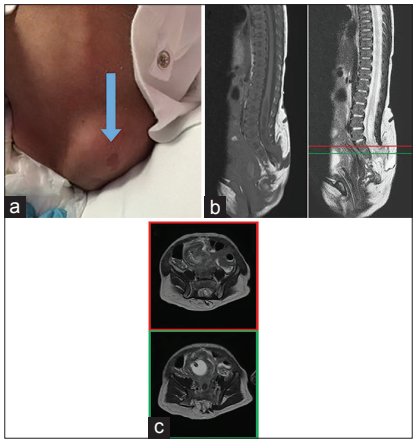

Figure 2: (a) Photograph demonstrating a lumbar mass and associated hyperpigmentation. (b) T1 (left) and T2 (right) sagittal sections of an MRI showing the lumbosacral dysraphism and associated cystic lumbar mass. (c) Selected T2 axial sections as identified by color coding on the sagittal image in A

the neural tube, which extends in both the rostral and caudal directions. The ectoderm overlying the neural tube separates to ultimately form the skin dorsal to the spine, a process known as dysjunction. Mesoderm around the neural tube differentiates into the posterior vertebral elements, fat, and paraspinal musculature. In premature dysjunction, mesoderm can migrate into the neural tube before it is fully closed, disrupting the neurulation process. This mesoderm then differentiates into fat and forms a border between the neural placode and the now entrapped lipoma. As development continues, meninges form around the neural tube except at the placode-lipoma interface, leaving a dorsal diaschisis traversed by a lipoma. This often results in a distinct transition point between normal planes and anatomy, anteriorly, and the lipoma, posteriorly.[13]

In secondary neurulation, a caudal mass of mesenchymal mesoderm cavitates and fuses with the primary neural tube, forming the spine below S2. After fusion of the primary and secondary neural tubes, mesoderm can migrate caudally and interfere with secondary neurulation in a mechanism similar to the disruption of primary neurulation. Secondary neurulation differs phylogenetically and is incompletely understood. Humans lack mature tail structure and have less complexity of secondary neurulation comparatively. In chick embryos, dynamic histology describes a coalescing of radially oriented tubules around a central lumen, ultimately cavitating within the caudal cell mass and joining the primary neurulated structure. Prevailing theories involve morphogenetic determinants, with candidate genes including sonic hedgehog and Pax transcription factors.[10] Environmental factors are posited to interfere with the enfolding mechanism of secondary neurulation including

folate deficiency, viremia, and teratogens as examples; however, little incidence data supports this supposition.[8]

The morphology of spinal lipomas is thought to be determined by which of the two neurulation processes is affected. Regardless of the morphology, in LMMC the placode-lipoma junction lies outside the spinal canal with dorsal extension of the meninges through an accompanying bony defect, in contrast to residing inside the canal, as seen in a lipomyelocele.[29] After the lipoma exits the dural defect, it continues through a fascial defect to communicate with subcutaneous tissue. This tethers the spinal cord and restricts its ability to ascend normally, making it susceptible to stretch injury during spine growth or repetitive ischemic insults resulting from flexion/extension movements.[15,21,24]

Classification

Traditionally, spinal lipomas have been classified into three groups based on the location of the neural placode–lipoma junction: Dorsal, caudal, and transitional, known as the Chapman classification[3,5] [Figure 3]. In dorsal spinal lipomas, the junction is on the dorsal aspect of the lumbar spinal cord and spares the conus medullaris. The dorsal root entry zone (DREZ) and neural elements are displaced lateral and ventrolateral to the placode–lipoma junction, respectively. The nerve roots emerge from the spinal cord tissue anterior to the junctional zone, where the lipoma, dura, and conus medullaris converge. In contrast, the conus is involved with caudal lipomas, and neural elements are located rostral to the junction. In caudal lipomas, the fatty tissue can extend from within the central canal caudally, where the fat is intermixed with nerve roots. The distal cord thus appears progressively larger in diameter toward the caudal aspect. Other findings may include a restrictive transverse fibrous band at the level of the last intact lamina. Transitional lipomas have characteristics of both dorsal and caudal types, with viable nerve roots passing through the lipoma tissue. These lesions tend to be asymmetric, with a rotational component on the spinal cord. The placode–lipoma junction is thus typically rotated. In addition, Pang *et al*. described a chaotic type, which has an irregular border between the placode and lipoma. Fat extends around the spinal cord and onto its ventral aspect, obscuring the DREZ. Most LMMCs are of the dorsal or transitional type.[30]

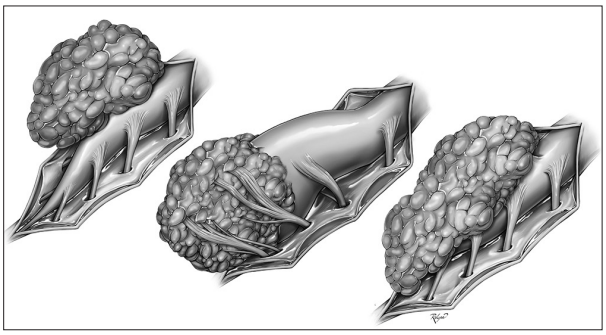

Figure 3: Classification of lipomas of the conus medullaris, as described by Chapman. Dorsal, caudal, and transitional types (right to left)

Premature dysjunction is necessary for all types of spinal lipomas, except the chaotic type. Primary and secondary neurulation is disrupted in the dorsal and caudal types, respectively, whereas both are affected in the transitional type. The chaotic type is thought to involve only secondary neurulation, where mesenchymal cells may become mixed with the caudal stem cell mass.

Presentation

Spinal lipomas and LMMCs are frequently associated with cutaneous and musculoskeletal abnormalities in addition to sensorimotor deficits and urological dysfunction.[29] Cutaneous lesions include subcutaneous lipomas, capillary hemangiomas, complex dimples, and hypertrichosis, whereas complex malformations, such as dermal appendages, are rare.[12,15] Musculoskeletal findings include scoliosis, unilateral or bilateral foot deformities, such as pes cavus, club feet, or abnormal rotation, or asymmetry of the foot or leg. Any of these findings should prompt consideration of an underlying embryomorphic etiology. Urological dysfunction, such as incontinence, frequency, urgency, and urinary tract infections, are also commonly associated. Neurological symptoms frequently correspond to those expected of a tethered cord syndrome, such as back or leg pain at rest that worsens with activity, in addition to weakness, sensory disturbances, or gait abnormalities.[9]

At birth, neurological symptoms may be absent in nearly half of the cases.[16] As the infant ages and axial growth occurs, the infant may experience progressive loss of neurological function.[19] Often, a change in the pattern of bladder and bowel function is the presenting symptom of LMMC.[9] As axial growth continues, lower limb, and sacral motor and sensory dysfunction, such as radicular pain, leg spasticity, foot deformities, and gait abnormalities, can develop.[17] Consequently, older children who escape early detection of LMMC are more likely to present with more pronounced urological and neurological complaints.[2] In addition to the symptomatic progression that correlates with axial growth, morphology of the defect also plays a large role in the presentation of LMMC patients. Symmetric malformations without a rotational component to the lipoma–placode interface tend to cause bilateral neurological or orthopedic abnormalities, which present at a later age. In contrast, asymmetric malformations tend to cause unilateral functional abnormalities and present earlier in life, usually on the side to which the neural placode was rotated.[2] Finally, LMMCs can become symptomatic from spinal stenosis secondary to

mass effect as the lipomatous malformation increases mass over time.[30]

LMMC can be associated with additional pathologies, including Chiari malformation type 1 (13%), spina bifida (14.4%), split cord malformations (3.1%), associated dermal sinuses (3.1%), dermoid or epidermoid cysts (3.1%), diastematomyelia (3.1%), terminal hydromyelia (3.1%), anal stenosis (1.0%), and Down syndrome (1.0%).[18,23,33]

DIAGNOSTIC STUDIES

Ultrasound is an effective screening tool because it is low risk and widely available, however, it has limited use after the initial diagnosis or following surgical treatment and should not be relied upon as the sole preoperative assessment.[22] A detailed MRI is the definitive imaging evaluation for spinal-neural lipomas. The anatomical detail of the placode-lipoma junction can be shown in relation to the normal spinal cord. Plain radiographs or computed tomography (CT) imaging may be useful to assess for scoliosis and evaluate the spine's bony anatomy during preoperative planning.

MANAGEMENT

Historical studies have shown that surgical interventions may briefly stabilize or relieve neurological symptoms but ultimately fail to improve upon the natural history of LMMCs.[6,21,23,28,34] Symptoms are typically progressive and worsen with age. Kulkarni *et al*. shed light on the natural history, citing a 33% risk of symptom deterioration with conservative management versus 46% for surgical treatment at nine-year follow-up.[28] One downfall of this study was that the conservative group was prospectively followed, whereas the surgical cohort was treated in the 1970s and retrospectively analyzed. Nevertheless, this study has changed the outlook and management of asymptomatic patients previously thought to require surgical intervention. The argument against surgical intervention for asymptomatic spinal lipomas was reinforced again in a 2012 London study that found a 40% cumulative risk of deterioration at 10 years.[35]

Surgical intervention may provide temporary relief or lessening of symptoms by releasing tension on the spinal cord, however, there is a risk of retethering with subsequent return or progression of neurologic symptoms, with reported rates of 5–50%. In a study by Colak *et al*. of 94 patients who underwent initial repair of a LMMC, 20.2% required subsequent operations for symptomatic retethering, with an average follow-up of 52 months after surgery. Of these reoperated patients, 6.4% exhibited repetitive symptomatic tethering, which became more difficult to treat and with shorter times between return of symptoms. Colak concluded that even after an adequate initial operation, symptomatic retethering is a common problem and that no current duraplasty graft material entirely prevents this from occurring.[7] There has been a confusing array of studies showing progression of neurologic symptoms after surgery to be lower,[4] similar to,[3,6] or worse than[11] the natural history. Cochrane *et al*. suggested that the variance in these results may be due to differences in the symptoms assessed, the duration of follow-up, the type of malformation, and timing of surgery.[6] Many experts argue for conservative treatment, with close clinical evaluations and surgical intervention only as patients develop worsening symptoms, as the natural history is similar or marginally better than the long-term outcome of surgically-treated patients.

A recent retrospective review attempted to identify radiological correlates predicting neurological decline. Over 16 years, a 24-patient population with LMMC that underwent an observational management strategy at a single institution was dichotomized into those experiencing early (less than 18 months) and late (18–30 months) neurological deterioration. Nine patients experiencing early deterioration were more likely to have large intradural lipomatous masses, which grew within the first year to exert regional mass effect on neural structures and were associated with a large expanded syrinx. Early decliners were more likely to present with motor deficits, whereas 15 patients experiencing late neurological decline presented with mixed urologic and motor deficits.[32] This study supports prophylactic untethering in infants with large intradural lipomas with syrinx, which exert mass effect on neural structures.

Several factors are thought to affect the treatment outcome of LMMCs. Age, gender, morphology, the presence and severity of neurological symptoms, and absence or presence of an associated spinal cord syrinx are all taken into consideration. Of these, morphology is considered the most crucial factor affecting outcome. For example, transitional lipomas appear to have a higher rate of retethering after surgery than dorsal and caudal types.[7] It is unclear if this is related to a factor intrinsic to the embryology or related to lesional complexity precluding adequate untethering.[30,31] It is also hypothesized that these factors may affect the outcome through a common pathway described by Pang *et al*. as the postoperative cord-to-sac ratio, which his group noted to be directly correlated to the progression or return of neurologic symptoms.[30] They suggested that cord retethering is a result of too little space in the dural sac for the spinal cord. This forces into close proximity any remaining residual lipoma surface with the normal spinal cord, which can then adhere. If the lipoma is completely removed and space within the dural sac is increased, they posited there is less chance of the cord contacting the dura or the "sticky" lipoma surface and retethering.

The traditional surgical technique described in historical studies involves partial resection of the lipoma to avoid injury to the neural placode, followed by untethering of the cord, then apposition of the edges of the placode, and finally duraplasty.[1] This technique, however, does not dramatically affect the cord-to-sac ratio. Pang *et al*. suggested a more aggressive approach involving total or near-total resection of the lipoma, complete reconstruction of the neural placode, followed by expansile duraplasty, preferably using bovine pericardium.[30] Using their technique, they showed no neurologic deterioration in 88.1% of patients over 20 years of follow-up, compared to 34.6% risk of progression over 10 years in patients with only partial resection. This included lipomas of all types, including symptomatic, asymptomatic, unoperated, and redo subgroups. Progression free survival (PFS) for asymptomatic unoperated spinal lipomas with this aggressive resection method was 98.8% over 20 years. Multivariate analysis of their data showed cord-to-sac ratio to be the only independent factor predicting outcome. These results have not been reproduced in any other series to date, however, they are compelling numbers for those who hold that surgical intervention can improve upon the natural history of LMMCs, whether or not symptoms are present.

A radically different approach to the treatment of tethered cord comes in the form of vertebral column shortening, which offers an alternative method for relieving tension on the spinal cord without risking injury to the neural placode and possibly stabilizing or improving neurological outcome.[20] The three-column osteotomy typically involves T12 or L1, and the average reduction in height is approximately 20 mm. Potential surgical complications include pseudoarthrosis, neurological injury, and significant perioperative blood loss. Kokubun *et al*. used this surgical method to treat eight patients (ages 15–54 years), 3 of which had previous conventional untethering procedures.[26] Six patients remained stable, and 2 patients experienced neurological decline over an average follow-up of 6.2 years. For these patients with decline, it is unclear if additional height reduction is needed, or perhaps changes within the neural placode occur independent of tension produced by the tethering. Although thought to be a relatively safe treatment option when used by experienced surgeons, its use should be limited to symptomatic patients with recurrent tethering despite conventional surgical detetherings, until studies with larger patient numbers can demonstrate its safety and effectiveness as a primary treatment option.

Regardless of the type of surgical intervention, the use of operative microscope is recommended, and intraoperative neurophysiological monitoring should be performed. There are a number of various neurophysiologic measures that can be used, and institutional practice may dictate what is available. Whatever monitoring type is chosen, the goal remains to avoid unintended injury to intact nervous structures, which may be hidden by or attached to the LMMC.[25,27]

As mentioned earlier, management of symptomatic LMMCs may not always entail surgical untethering. Complex variants of the transitional or chaotic spinal lipomas associated with isolated urological symptoms or orthopedic deformities may be observed because these abnormalities are less likely to improve with surgical intervention. They also carry a higher risk of unsuccessful untethering and incomplete lipoma resection, which may lead to an increased risk of neurological deterioration after surgery.

After careful evaluation, "simple" LMMCs with symptoms or any LMMC with an associated sensorimotor deficit should be considered for possible surgical intervention. Caudal and dorsal spinal lipomas are typically more amenable to surgical treatment than the rest. Immediate postoperative complication rates range from 10–30% and include infection, CSF leak, or neurological deterioration.[1,23]

LMMCs associated with embryomorphic malformations of other systems may represent the more severe end of the spectrum of congenital defects. For example, OEIS (omphalocele, exstrophy, imperforate anus, spinal defects) and VATER (vertebral defects, anal atresia, tracheoesophageal fistula, renal abnormalities) represent associated multisystem abnormalities, which complicate management and are beyond the scope of this review.

CASES IN CONTEXT

Table 1 summarizes the salient details of Cases 1 and 2, along with the resultant clinical decision. Case 1 included incidental identification of an asymptomatic caudal LMMC with associated syrinx and without intradural mass effect. The patient had normal motor and urological exams, with only the development of leg tightening over 2 months. The clinical and radiographic data were combined into a clinical decision rule supported by the literature, and the patient has been stable through

Table 1: Clinical decision-making for patients with lipomyelomeningocele

	Clinical data	Radiographic data	Management decision
Case 1	Incidental identification	Small syrinx, no mass effect	Observational treatment
Case 2	symptomatic, static or worsening deficits	Large syrinx and mass effect	Debulking and untethering

observational treatment. Case 2 is defined radiographically by caudal LMMC with large associated syrinx and mass effect on intradural neural structures. The patient also exhibited signs of tethering, including diminished deep tendon reflexes and abdominal distention, along with intercurrent embryomorphic malformations of the spine and renal system. In the presentation for Case 2, the literature supports untethering and debulking, which was performed. The patient had essentially static symptoms without significant worsening or improvement on follow-up at 18 months.

CONCLUSION

LMMC management remains a challenge. The selected cases demonstrate important factors integrated within a clinical decision rule. Although there is no high-quality clinical outcome data to provide guidance regarding the treatment options for LMMCs, conservative management of asymptomatic patients is appropriate. Clearly progressive symptomatic patients should be considered for surgical untethering with the goal of managing symptoms, with the patient and family prepared for an iterative process. Patients with static neurological deficits should be managed observationally. Prophylactic surgery may, theoretically, prevent the onset of neurological deterioration or stabilize and reverse early-onset symptoms at diagnosis, especially in infants with large intradural lipoma and associated syrinx, which compress neural structures, however, this has not been shown to offer immunity against further deterioration. When surgical management is elected, experts advocate aggressive resection of the lipoma, along with reconstruction of the placode and large expansile duraplasty, but the literature shows this is technically difficult and may not greatly improve upon the natural history of LMMCs.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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