Intradural mesh: an unusual cause of spinal cord tethering

Case report

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A 13-year-old boy with a myelomeningocele experienced progressive foot deformity and lower-extremity pain while walking. Magnetic resonance imaging demonstrated a low-lying spinal cord with scarring near the site of a previous repair. During surgery, the terminal nerve roots were found to have scarred and adhered to a piece of metal mesh lying in the intradural space. The mesh had originally been placed to bridge a sacral ossification defect that was present at the initial closure of the child’s myelomeningocele.

KEY WORDS • tethered spinal cord • tethered cord syndrome • myelomeningocele • spina bifida • pediatric neurosurgery

Perhaps 15 to 30% of patients with a myelomeningocele will experience symptomatic tethered cord at some point during their lives. The most common cause of this disorder is intradural scarring at the site of the initial closure. Other lesions that are recognized to cause tethering in this patient population include dermoid or epidermoid cysts, thickened filum, diastematomyelia, neurenteric cysts, and lipomas. We describe a child with a tethered cord that was caused by dense adhesions between terminal neural tissue and a small piece of metal mesh that had been used for sacral closure and had, subsequently, migrated into the intradural space.

Case Report

History. This 13-year-old boy presented with a progressive foot deformity and lower-extremity pain while walking. Before his birth a lumbosacral myelomeningocele had been recognized with the aid of prenatal ultrasonography, and the lesion was repaired on the 1st day of the patient’s life. As part of the closure, a small (3 × 1.5-cm) piece of metal mesh was used to bridge a sacral bone defect (Fig. 1). The mesh was secured in a plane lying deep to the lumbar dorsal fascia. Recovery from this operation was uncomplicated. A ventriculoperitoneal shunt was placed a few days later. The child had done well up to the current presentation.

The patient was initially examined at our institution when he was 10 years old. He was receiving instruction in a regular classroom and was able to walk without assistive devices, although he did have slight, stable weakness of his ankle everters. At that time MR imaging revealed bone defects at L-4, L-5, and in the sacrum. The conus medullaris ended at the S-2 level. There was a small lipoma at L-4 and a thin syrinx that extended from T-9 to S-2.

Examination. When the boy was 13 years of age, he presented to our clinic complaining of pain in his left leg. We found new foot deformities and were told he had recently experienced several urinary tract infections. Another MR image was obtained, but the findings were not significantly different from those observed 3 years earlier (Fig. 2). Because of his symptoms, we decided that the boy should undergo a spinal cord detethering procedure.

Operation. During surgery, the terminal nerve roots were found to have scarred and adhered to a piece of titanium mesh located in the intradural space (Fig. 3). Several roots were stuck to the mesh, and a few rootlets seemed to traverse the holes in the mesh. The roots were separated from the metal and it was removed. The dura mater was closed.

Postoperative Course. The patient had an uneventful postoperative course and was discharged without new deficits. Eight months postoperatively, he has no leg pain and his foot deformity is stable.

Discussion

Spinal cord tethering is a major problem for patients with a myelomeningocele. It occurs frequently and can lead to
substantial neurological disability. Operative treatment can be technically demanding and there is a 10 to 15% chance of recurrence. The most common cause of tethering is scarring at the site of the initial closure. Most surgeons think that the severity of scarring and, perhaps, the likelihood that a patient will experience tethered cord syndrome are related to the operative technique that is selected.3,8 Consequently, a number of surgeons have modified the standard techniques used for myelomeningocele closure in the hopes that their changes will minimize the formation of adhesions between the neural placode and the overlying dura mater. It is thought that this can best be accomplished by preventing

![Fig. 1. Lumbar x-ray film demonstrating a piece of metal mesh located at the midline as well as bone defects.](image1)

![Fig. 2. Magnetic resonance images revealing tethered nerve roots and a low-lying conus. Note the minimal artifact associated with the titanium mesh. This is a characteristic of the material, pointed out in the paper by Sullivan, et al.](image2)

![Fig. 3. Intraoperative photograph showing the pieces of metal mesh in an intradural position.](image3)
compression of the placode and ensuring that there is a large dural sac with cerebrospinal fluid surrounding the cord within the tube.3,8

In the case that we describe, a piece of titanium mesh was used to reconstruct the posterior bone canal at the site of the dysraphic defect. In the original operative report the surgeon had noted the use of the mesh but did not explain its purpose. Presumably, the mesh was used to protect the cord and prevent its compression while also allowing a relatively large space for the dural sac. Over time, however, the mesh migrated into the intradural space. Rather than prevent the cord from tethering, the mesh became a component of the tethering lesion.

This situation is similar to the intracranial migration of rigid fixation devices. When metal plates and screws are used to repair the cranial vault in young children, these devices can become incapsulated by bone growing around them. As the skull grows new bone is laid down outside the hardware and the bone beneath it is resorbed. Occasionally, this results in hardware within the cranial vault.2

In the patient whom we describe, it is unclear if the mesh was incorporated into the margins of the sacral defect and migrated through them or if the mesh became loose as the sacrum grew and was left unsupported within the bone defect. In either case, once the mesh was free from the bone it was incorporated into the dura mater and, ultimately, came to lie in the intradural space, leading to the patient’s neurological decline and the need for another operation.

It is possible that the choice of materials contributed to the problem. Metal mesh is more likely to form cell adhesion complexes than some other implantable materials such as ceramics. In addition, titanium is more prone to the formation of these complexes than other metals.5 Perhaps this propensity led to the dural incorporation of the mesh.

The use of mesh to reconstruct the bone canal during the closure of a myelomeningocele is theoretically attractive. Unfortunately, there is a risk of plate displacement as the child grows. Although the present case does not allow quantification of that risk, experience with cranial rigid fixation in very young children would lead us to infer that the problem is potentially significant.

References


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