Recurrent spinal cord tethering by sacral nerve root following lipomyelomeningocele surgery

Case report

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A 21-year-old woman had recurrent progressive weakness/hypesthesia and pain in both lower extremities. At the age of 5 and 19 years, she had undergone surgical resection of a lipomyelomeningocele at L5–S1. Surgical exploration revealed that the cord was tethered and pulled over to the side by an excessively short right S-1 nerve root. The contralateral L-5 and S-1 nerve roots were markedly stretched. Division of the right S-1 nerve root resulted in prompt disappearance of pain in the lower extremities and improvement in neurological function.

KEY WORDS • tethered spinal cord • nerve root • lipoma • meningocele • spinal cord

Tethered spinal cord is a relatively uncommon condition. Tethering mechanisms that have been described include a short filum terminalis, lipomas of the cauda equina/conus medullaris, and adhesions or fibrous bands tethering the conus to the dura or surrounding structures. We describe a case where repeated tethering occurred following two procedures for removal and repair of lipomyelomeningocele. The tethering, which caused progressive neurological deficit and intractable pain in the lower extremities, was due to an excessively short S-1 nerve root on the right side. Division of the nerve root provided excellent relief from the symptoms.

Case Report

This 21-year-old woman presented with the complaint of progressively increasing pain and neurological deficit in the lower extremities from possible recurrent lipomyelomeningocele. At the age of 5 years, she underwent resection of a sacral lipomyelomeningocele elsewhere. At that time her neurological deficits consisted of flaccid paralysis of the bowel and bladder function as well as weakness in the distribution of the lower lumbar nerve roots. At 19 years of age, a recurrent lipoma was surgically removed. Approximately 1 year after the procedure, the patient noticed recurrence of severe pain in the lower extremities. The pain radiated along the posterior aspect of both lower extremities. She also noticed progression of weakness and hypesthesia in the left L5–S1 distribution.

Examination. Neurological examination at the time of our initial assessment showed marked bilateral weakness of the muscle groups in the L4–S1 distribution. The anal sphincter was flaccid. There was complete anesthesia in the perineal area, anesthesia in the right S-1 distribution, and hypesthesia in the left S-1 and bilateral L-5 distribution. Straight-leg raising elicited severe diffuse pain in both lower extremities. No definable evoked lumbar or thoracic responses to peroneal or tibial nerve stimulation could be identified on either side. Magnetic resonance (MR) images of the cervical and thoracic spine were normal. An MR study of the lumbar area showed that the spinal cord ended at L-5. There was a large meningocele at L5–S1, but there was no evidence of recurring tumor. An iohexol myelogram followed by a computerized tomography scan showed the spinal cord at the L4–5 level to be small and deviating toward the posterior and right aspects of the dural sac. Because of relentless progression of the signs and symptoms, it was decided to re-explore the lumbar area.
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The cord was found to be displaced and pulled over to the right side. The right S-1 nerve root was smaller than normal and was under extreme tension (Fig. 2 left). The left S-1 nerve root, which was made up of about six or seven rootlets, was markedly stretched and under extreme tension (Fig. 2 center). The left L-5 nerve root was also under moderate tension due to the displacement of the cord. The right S-1 nerve root was stimulated electrically, and plantar flexion of the right foot was elicited. There were no intradural adhesions constricting or tethering the S-1 root. It was clear that the S-1 nerve root was responsible for the deviation of the spinal cord and the tension on the contralateral roots. Clinically, the patient had minimal function attributable to the right S-1 root. The nerve root was divided, and immediately the cord moved to a midline position with most of the tension and stretch relieved from the contralateral roots (Fig. 2 right). The dura was closed in a watertight fashion and the postoperative course was uneventful.

**Postoperative Course.** The patient immediately noticed complete relief from the bilateral lower-extremity pain. In the 2 months following the procedure she also noticed further improvement in strength and sensation in the S-1 and L-5 dermatomes on the left side. She returned to her previous employment 1 month after the procedure. At her 1-year follow-up examination, the positive results persist.

**Discussion**

This patient had symptoms and signs of persistent and progressive tethering of the spinal cord/nerve roots...
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following two procedures for lipomyelomingecele removal and cord untethering. Several mechanisms can account for recurrent tethering: recurrence of the lipoma, incomplete initial division of the filum terminais, or further tethering of the cord and filum by scar tissue or adhesions. The cord can also become tethered to the dura if an appropriate dural closure was not performed.²

This case demonstrates that, even in the presence of a completely untethered or absent (as in this patient) conus medullaris and even with no adhesions constricting the cord or tethering it to the dura, severe traction can still exist on the nerve roots/spinal cord. When our patient underwent the second surgical procedure, her growth spurt was already over. The tethering on the lumbar roots was already present and removal of the recurrent lipoma did not relieve it.

In a review of 18 patients who, as adults, suffered the onset of spinal cord tethering, Pang and Wilberger¹ found that pain was the most common symptom, being present in 80% of the cases. This is in contrast to the syndrome with childhood presentation, where pain is seldom present. They acknowledged that the origin of the pain is uncertain but attributed it to some central mechanisms, possibly to the tension exerted on the conus medullaris. They stated that traction on the nerve roots of the cauda equina is seldom a mechanism, since these roots are usually not under tension.

In our case, pain was clearly related to the extreme degree of tension of the L5–S1 nerve roots. This also agreed with the clinical signs and symptoms of nerve root irritation. Since pain, which is increased by mechanical stretching of the nerve roots, seems to be a frequent complaint in tethered cord syndrome of adult onset, it is possible that tension on the nerve roots might be an overlooked mechanism. Since our patient already had an advanced long-standing dysfunction of the right S-1 nerve root, the decision to divide it was an obvious one. Had the deficit been milder or of more recent onset, the decision on what course of action to take could have been more difficult. This is one situation where, in order to prevent further damage to the adjacent nervous structures, one has to sacrifice certain neural elements.

References


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