Spasticity Management with Intrathecal Baclofen Complicated by Syringomyelia in a Patient with Spastic Paraplegia

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Patient Setting

Setting: Outpatient Spinal Cord Injury (SCI) Clinic
Patient: A 35-year-old male with a history of insulin dependent diabetes mellitus, diabetic peripheral neuropathy, and spasticity secondary to a traumatic T6 ASIA A SCI approximately 13 years prior managed with an intrathecal baclofen (ITB) pump.

Learning Objectives

1. Illustrate the role of complications secondary to syringomyelia in the management of chronic spinal cord injury.
2. Promote discussion of intrathecal baclofen management and troubleshooting in the management of chronic spinal cord injury.
3. Differentiate common and uncommon causes of worsening spasticity in the management of chronic spinal cord injury.

Post-Traumatic Syrinx After Spinal Cord Injury

Syringomyelia involves the formation of a cavity, termed syrinx, within the spinal cord. This expands in a rostral and caudal direction longitudinally, and in diameter. This may result in neurologic deterioration. Studies of SCI patients 1 – 30 years after injury have demonstrated that around 20% will develop a syrinx[1]. It is estimated that up to 8% of patient with SCI may develop symptomatic progressive myelopathy[1]. The most common clinical presentation is pain. Currently, MRI remains the gold standard for diagnosis and follow-up of post-traumatic syrinx (PTS).

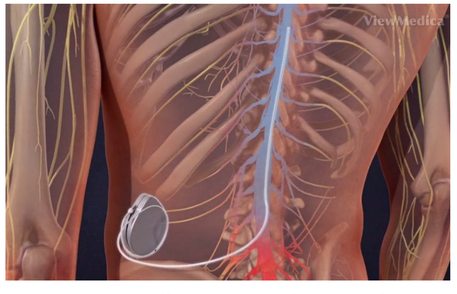

Figure 1. Intrathecal Pump System *http://summitpainfw.com/services-view/pain-pumps/*

Case Timeline

- The patient presented to SCI clinic with worsening spasticity after several years of satisfactory management with intrathecal baclofen (ITB). Pump dosing was increased, and as needed valium was prescribed.
- Subsequently, he identified the spasticity being worse when lying down. Dosing was increased over several visits and a bolus dose was programmed.
- Due to no improvement with increased baclofen dosing, or bolus response in the setting of a otherwise negative workup an MRI with contrast was ordered.
- The MRI spine with contrast demonstrated a mid-thoracic syrinx from T5-T9.
- Neurosurgery was consulted, but did not offer operative intervention with regard to the syrinx.
- The patient continued to have poorly controlled spasticity despite pump dosing increases and addition of oral tizanidine.
- A catheter study was performed that was negative for catheter flow abnormalities.
- A pump replacement was performed with catheter revision.
- Patient continued to have increased spasticity. Additional catheter study demonstrated flow restriction near the catheter tip, and patient returned to surgery for pump and catheter replacement with tip of the catheter placed at T4.
- At follow-up, his spasticity was unchanged. Repeat MRI demonstrated a stable syrinx.
- Due to further recalcitrance to spasticity management the catheter tip was withdrawn to T10 due to the presence of syrinx.
- The patient's pump dosing was titrated incrementally and clonidine was added for neuropathic pain in the setting of diabetic polyneuropathy with good response.
- At subsequent follow-up, the patient noted return to baseline spasticity.

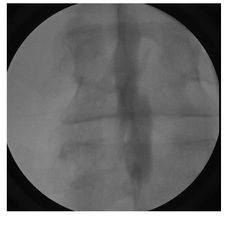

Figure 2. Fluoroscopic image of patient's patent catheter study

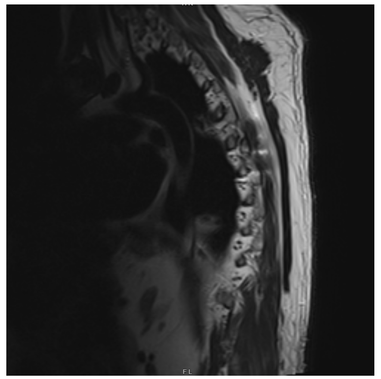

Figure 3. T2 weighted sagittal MRI of the patient's thoracic spine demonstrating syrinx

Conclusion

This case elucidates a unique complication of PTS in SCI. The patient's presenting symptom was positionally aggravated worsening spasticity in the setting of an otherwise negative workup. Subsequent MRI and catheter studies failed to demonstrate complications consistent with the patient's symptoms. Only after withdrawal of the catheter below the level of the syrinx did the patient's symptoms improve. This demonstrates the need to consider CSF flow restriction secondary to a PTS in patients managed with an ITB pump and worsening spasticity.

References

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