

## The problematic presence of post-traumatic syringomyelia when new weakness develops in chronic cervical SCI

Gretchen A. Ferber, MD, MFA<sup>1</sup>, Kristen A. Milleville, MD<sup>1</sup>, Amanda L. Harrington, MD<sup>1</sup>

<sup>1</sup>University of Pittsburgh Medical Center, Department of Physical Medicine and Rehabilitation

### Background

In patients with chronic cervical spinal cord injury (SCI), any neurological decline may significantly alter function. Therefore, etiology of new weakness must be promptly and accurately diagnosed. An uncommon complication of chronic SCI is post-traumatic syringomyelia. While the typical clinical symptoms of syringomyelia involve gradual loss of motor function, cases of acute onset symptomatology exist in the literature (Bodley, Jamous). The incidence of post-traumatic syringomyelia varies widely, occurring in <1 to 7% of patients with chronic spinal cord injury with radiological findings ranging from 20 to >50% (Bodley, Chen, Li). Compressive peripheral neuropathies among chronic SCI patients have been widely described in the literature with many areas of possible compression depending upon the patient's functional level (Bursell). Unlike neurologically intact individuals, diagnosis of peripheral nerve injuries in those with chronic SCI is confounded by pre-existing neurological deficits (Hitchman). In a patient with chronic SCI with new neurological deficits, diagnostic work-up includes imaging of the spine with CT myelography and/or MRI. With a discordance of patients with symptomatic post-traumatic syringomyelia compared to those with radiographic findings, it is likely a large portion of patients with post-traumatic syringomyelia are, in fact, asymptomatic. Therefore, presence of syringomyelia on imaging may complicate a work-up in the setting of new neurologic deficits.

### Case Details

Case 1: 32-year-old male with history of chronic traumatic SCI, C5 American Spinal Injury Association Impairment Scale (AIS) B.

- 8 years post-injury, patient at high functional level: living independently, working, playing wheelchair rugby, driving with hand controls
- Experienced acute-onset left wrist and forearm weakness
- MMT in left wrist extensors 3/5, previously 5/5
- MRI cervical spine initially found increased fluid intensity at C5, concerning for syrinx
- CT myelogram of cervical, thoracic, and lumbar spine found atrophy and distortion of spinal cord at C5 without fluid collection
- EMG/NCS, 9 days after onset of symptoms, found chronic, incomplete left C6 radiculopathy, with a structurally and mechanically intact left radial nerve on electrodiagnostic studies and ultrasound
- One month later, patient had resolution of weakness without intervention

### Case Details, cont.

Case 2: 21-year-old male with history of chronic traumatic SCI, C5 AIS A and traumatic brain injury.

- 3 years post-injury, patient awoke with acute onset left wrist weakness with loss of tenodesis
- MMT revealed 1/5 strength in left wrist extensors, previously 5/5
- MRI cervical spine found 5 x 12 mm mildly expansile central cystic or syrinx cavity at the level of C5 and C6
- Neurosurgery was consulted and recommended no immediate surgical intervention
- Repeat MRI cervical spine was obtained one month later, and the size of the syrinx was stable
- Patient's strength returned over the next 4 months, with MMT revealing 4/5 strength in left wrist extensors at four months; as such, he declined recommended follow-up studies of EMG/NCS and CT myelogram



Figure 1. Case 1: Sagittal view of CT myelogram of the cervical spine

### References

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### Discussion

In both patients, acute onset, asymmetric weakness was observed, primarily affecting the wrist extensors, resulting in significant functional decline. Both patients had no known history of post-traumatic syringomyelia at baseline.

Case 1 received a full work-up, including MRI cervical spine, CT myelogram of the entire spine, and EMG/NCS. While patient's clinical presentation was suspicious for radial nerve palsy, electrodiagnostic and ultrasound findings did not support the diagnosis. Although initial MRI findings were concerning for C5 syrinx, CT myelogram did not support these findings. Patient had resolution of weakness without surgical intervention within one month, which is atypical for syrinx presentation. Additionally, electrodiagnostic studies effectively ruled out peripheral nerve injury as an alternative diagnosis.

Case 2 had a work-up including MRI of the cervical spine, which confirmed the presence of a syrinx at C5-C6. Incidentally, patient had a repeat MRI of the cervical spine one month later during an admission for respiratory insufficiency, which found stable syrinx size. Patient's strength improved over the course of 4 months, and additional diagnostic studies were not pursued.

These cases demonstrate the diagnostic quandary of new weakness in the presence of or concern for post-traumatic syringomyelia on imaging. In the absence of baseline imaging, it is challenging to determine if timing and course of post-traumatic syringomyelia align with clinical symptoms of new weakness. Electrodiagnostic studies are an important adjuvant to imaging studies in these cases to assess for peripheral mononeuropathy.

### Conclusion

These cases illustrate the diagnostic quandary of determining if the presence of a post-traumatic syringomyelia on imaging explains clinical changes that may occur after cervical SCI. When evaluating new neurologic deficits, clinicians must decide upon diagnostic testing to determine the most likely etiology and prescribe a treatment plan. Because post-traumatic syringomyelia may contribute to clinical deficits, clinicians should consider obtaining baseline imaging after the initial traumatic injury to document the presence or absence of syringomyelia, which could help with future diagnostic dilemmas.