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ACUTE HEMIPARESIS AS THE INITIAL MANIFESTATION OF CHIARI I-ASSOCIATED SYRINGOMYELIA: A STROKE MIMIC

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ABSTRACT

Introduction: Syringomyelia is a rare neurological disorder characterised by the formation of a fluid-filled cystic cavity (syrinx) within the spinal cord, most commonly resulting from disruption of cerebrospinal fluid flow. It typically presents as slowly progressive dissociated sensory loss in a cape-like distribution involving bilateral shoulders, upper back, neck and arms, and is often accompanied by muscle weakness.

Case description: A 44-year-old female presented with sudden-onset posterior headache followed by right-sided blurred vision, right-sided hemiparesis along with numbness and paraesthesia. The initial evaluation ruled out a stroke. Magnetic resonance imaging (MRI) of the brain and spinal cord revealed an Arnold Chiari-I malformation with an extensive multiseptate syrinx from the cervical spinal cord to the conus medullaris. Multiple teleneurology consultations with multiple repeat imaging failed to elucidate the cause of the patient's progressive symptoms. Persistent and progressive neurological deterioration prompted a transfer for an in-person neurology evaluation where the definitive diagnosis was made, resulting in urgent transfer to a tertiary level centre for emergent posterior fossa decompression.

Conclusion: This case highlights an unusual presentation of syringomyelia with acute, rapidly progressive unilateral neurological deficits without an identifiable precipitating event, closely mimicking acute ischaemic stroke. It emphasises the need to maintain a broad differential diagnosis when the presentation is atypical, or investigations do not explain the neurological deficits. This case also underscores the importance of comprehensive bedside neurological examination, as reliance on teleneurology may delay diagnosis. Early recognition and timely neurosurgical intervention can result in substantial neurological recovery.

KEYWORDS

Chiari I-associated syringomyelia, acute hemiparesis, stroke mimic, cord compression, neurological emergency

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LEARNING POINTS

- Syringomyelia should be considered in the differential diagnosis of patients presenting with atypical unilateral neurological deficits when imaging studies are non-conclusive for stroke.
- Careful neurological examination remains crucial in distinguishing spinal cord disorders from cerebral causes of weakness.
- Syringomyelia presenting with acute, rapidly progressive neurological deficits and spinal cord compression requires urgent neurosurgical evaluation and intervention.

INTRODUCTION

Syringomyelia is a rare, often slowly progressive neurological disorder characterised by the development of a fluid-filled cavity (syrinx) within the spinal cord. Syringomyelia is classified as either congenital, most commonly associated with Arnold Chiari-I malformation (CM-I), or acquired secondary to spinal cord tumours, arachnoiditis, subarachnoid haemorrhage and spinal cord trauma[1].

The prevalence of syringomyelia was estimated at 3.3 to 8.5 per 100,000 individuals before modern neuroimaging and 1.9 to 8.4 per 100,000 in the MRI era with increased detection of incidental and asymptomatic cases[2]. The disease typically follows a chronic course over months to years, seen in patients between 20 and 40 years of age[3]. The disease classically presents as pain and temperature insensitivity, radicular pain, gait ataxia, dysesthaesia, motor weakness and spasticity[4]. Additionally, acute presentations attributed to syringomyelia are typically preceded by a provoking event such as trauma or sudden Valsalva manoeuvres such as when coughing, sneezing, straining or heavy lifting[5].

This case is notable for its atypical acute onset in the absence of a provoking factor and rapidly progressive unilateral neurological deficits which initially mimicked an acute ischaemic stroke. Such presentations are exceedingly uncommon and may delay diagnosis and timely neurosurgical intervention. This case highlights an unusual presentation of Chiari I-associated syringomyelia and emphasises the importance of correlating progressive neurological findings with spinal imaging when the initial stroke evaluation is unrevealing. This case provides an overview of the established causes, underlying pathophysiology and management strategies of syringomyelia while underscoring a rare, unusual presentation and discussing plausible mechanisms responsible for its deviation from the typical clinical course.

CASE DESCRIPTION

A 44-year-old female with no significant past medical history presented to the emergency department with sudden-onset headache, right-sided neck pain, weakness involving the right upper and lower extremities, accompanied by tingling and numbness that started 45 minutes prior to presentation. She scored four on the National Institute of Health Stroke Scale (NIHSS). Computerised tomography (CT) of the head without contrast showed no acute abnormality. Neurology consultation was completed via the Telemedicine service for a suspected acute stroke. The patient declined Tenecteplase (TNK) therapy. Laboratory work-up including a complete blood count, a comprehensive metabolic panel, lipid profile, thyroid function tests, c-reactive protein, troponin and vitamin B12, which were all normal. A CT angiogram of the head and neck was performed, which was negative for large vessel occlusion, stenosis or aneurysm. The patient was diagnosed with presumed hemiplegic migraine and admitted to the hospital for further work-up. An MRI of the brain without contrast showed no evidence of acute infarct or haemorrhage, but did indicate

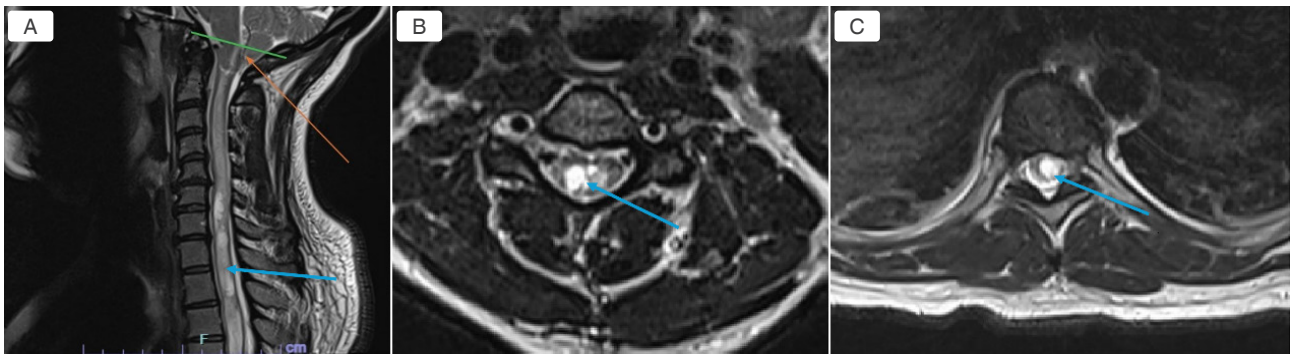


Figure 1. *Presurgical T2-weighted MRI images - Chiari-1 malformation, syrinx. A) Sagittal view of cervical spine demonstrating Chiari-1 malformation (orange arrow) and syrinx (blue arrow). Green line indicates the level of foramen magnum; B) Axial view of cervical spine demonstrating multiseptated fluid filled cavity consistent with syrinx (blue arrow); C) Axial view of thoracic spine at T5 level with blue arrow demonstrating extensive syrinx with associated cord compression.*

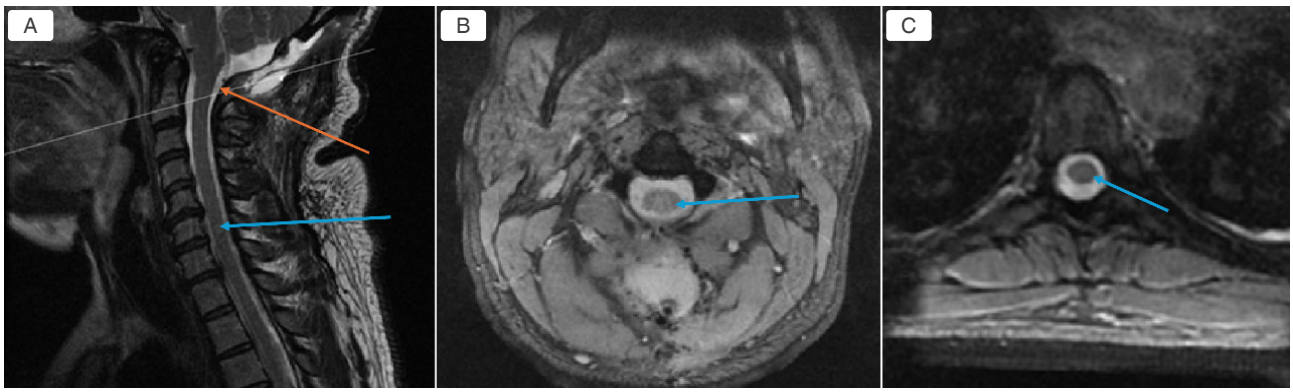


Figure 2. *Postsurgical T2-weighted MRI images – resolution. A) Sagittal view of cervical spine demonstrating successful Chiari-1 decompression (orange arrow) with complete resolution of syrinx (blue arrow). Grey line indicates the level of foramen magnum; B) Axial view of cervical spine demonstrating resolution of syrinx (blue arrow); C) Axial view of thoracic spine at T5 level demonstrating resolution of syrinx (blue arrow).*

long segment T1 hyperintense and likely T2 hyperintense signal abnormality involving the right cervical spinal cord. A dedicated MRI of the cervical spine with and without contrast was ordered for further clarification, which showed multiseptate cervical and thoracic syrinx. Cerebellar tonsils extend 12 mm below the occiput compatible with Chiari I malformation as seen in *Figures 1 A and 1B*.

Forty-eight hours after hospitalisation, the patient's weakness and numbness slightly worsened, which shed doubt on the hemiplegic migraine diagnosis. She developed diminished sensations in her groin area and new urinary retention. When the patient was re-evaluated by teleneurology, an MRI on the cervical spine was repeated and was unchanged. A dedicated MRI of the thoracic spine with and without contrast was ordered that showed extensive multiseptated thoracic cord syrinx up to the conus medullaris with maximal transverse dimensions approximately 9 × 9 mm in the upper and mid thoracic spine was noted as seen in *Figure 1C*. The case was discussed over the telephone with Neurosurgery, however as no in-person neurologic examination had been done, only the examination findings from the NIHSS were discussed therefore Neurosurgery felt the symptoms did not match with MRI findings and recommended outpatient follow-up.

After 72 hours of hospitalisation the patient noted hyperalgaesia on the entire right side of the body along with worsening right-side weakness. She had additional symptoms of right-sided stabbing chest pain. A CT of the chest and the shoulder without contrast were negative. Teleneurology was once again consulted. Another MRI of the brain with and without contrast was negative, and it was recommended that the patient was transferred for an in-person evaluation by a neurologist.

Upon a detailed neurological examination, she was found to have a normal examination on the left. On the right she had proximal greater than distal weakness with 2/5 strength on the medical research council (MRC) muscle strength grading scale in the deltoid, triceps, biceps and wrist muscles, and 3/5 strength in the fingers. The same pattern of weakness was noted in the legs with 2/5 weaknesses at the hips and knee but 3/5 with foot testing. She had decreased temperature and vibration sensation on the right and was also exquisitely painful on the right. Light touch was intact; however, due to her extreme pain on the right it was not possible to access symmetry. Her reflexes were reduced at the right biceps. Based on the neurologic examination and the visual observation that her cord displacement was worst at T5 and this correlated with the location of the patient's chest pain it was determined that her syrinx was symptomatic. She was transferred to a tertiary care centre for neurosurgical evaluation due to worsening symptoms concerning cord compression secondary to syringomyelia. At the accepting facility, the patient underwent urgent suboccipital craniectomy, C1 laminectomy, duraplasty and reduction of intradural cerebellar ectopic tissue for Chiari malformation. Following the procedure, the patient's right upper and lower extremity strength improved to 4+/5 and 5/5 on the lower extremities. Right-sided hyperalgaesia also improved and urinary retention was resolved; she had slightly diminished light touch on the right. The patient was discharged home with a walker and home rehabilitation services five days after the surgery. A follow-up MRI of the spine two months later showed significant improvement of the syringomyelia as seen in *Figures 2A, 2B* and *2C*. A chronological summary of the patient's clinical course is presented in *Figure 3*.

DISCUSSION

The most unusual feature of this case was the acute unilateral onset of symptoms. The patient's presentation initially raised concern for an acute stroke; hemiplegic migraines were also considered in the differential diagnosis. However, serial CT and MRI imaging demonstrated no evidence of cerebral infarction, and hemiplegic migraines became less likely due to worsening of her right-sided weakness over days. Her decreased vibration and temperature sensations as well as asymmetric reflexes were not discovered until she had an in-person neurologic examination.

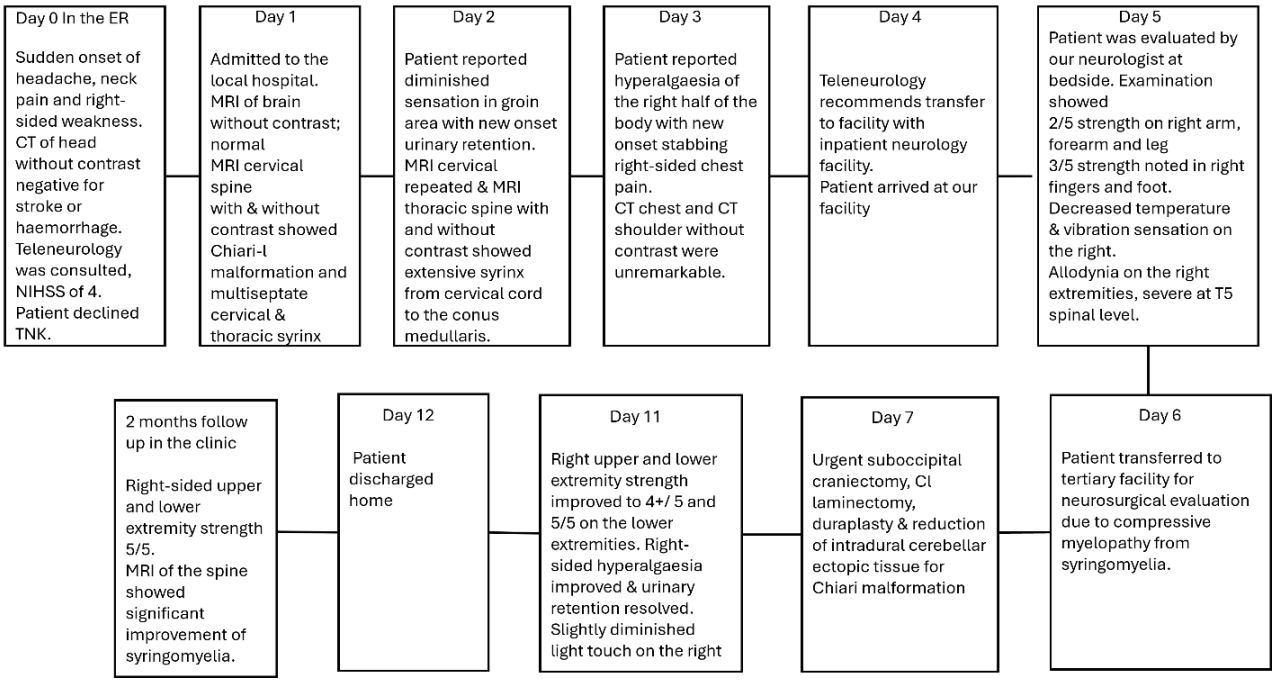


Figure 3. *Flow chart illustrating the chronological sequence of the patient's clinical presentation, diagnostic evaluation, treatment and clinical outcome.*

The classic presentation of syringomyelia is characterised by dissociated sensory loss, a pattern in which pain and temperature sensations are impaired while vibration and proprioception are preserved. This occurs due to the slow expansion of a central syrinx within the spinal cord which initially damages the anterior white commissure. This region contains decussating spinothalamic tract fibres responsible for transmitting pain and temperature sensation. In contrast, the dorsal columns which carry vibration, proprioception and fine touch are typically spared in the early stages of the disease[6]. Bladder and bowel dysfunction represent the late stage of spinal cord dysfunction.

The mechanism responsible for acute, unilateral onset of symptoms is that rather than remaining confined to the central canal, the syrinx had expanded sufficiently to produce a significant mass effect, compressing and displacing the spinal cord to the right along with the involvement of the dorsal columns. This distortion of the normal spinal cord anatomy provides a plausible explanation for the unilateral neurological deficits.

A similar case of syringomyelia causing acute hemiparesis was described by Frogameni et al.[7], which occurred in a football player following an injury while lifting heavy weights and symptoms resolved with conservative management. Our patient has a similar presentation but there were no identifiable immediate provoking factors preceding the onset of symptoms, and she required immediate surgery due to worsening symptoms.

In another case described by Ribeiro et al.[8], a 51-year-old female who presented with sudden loss of consciousness after progressive decrease in extremity strength was found to be in acute respiratory failure due to cervical syringomyelia from CM-I and needed immediate surgical intervention. This case has a similar acute presentation to our patient with acute symptom onset in the absence of a provoking factor.

Management of Chiari I-associated syringomyelia should be guided primarily by the patient's neurological status rather than the radiographic extent of the syrinx alone[6]. Although many patients with asymptomatic or minimally symptomatic syringomyelia can be managed conservatively, rapidly progressive neurological deficits constitute a neurosurgical emergency[9], because delayed decompression may result in irreversible spinal cord injury. Posterior fossa decompression with duraplasty remains the standard surgical treatment[10] and is associated with favourable neurological recovery in most symptomatic patients. Our patient's marked improvement following surgery further emphasises the importance of timely recognition and intervention.

Acute hemiparesis secondary to Chiari I-associated syringomyelia is an exceptionally rare stroke mimic. In patients with persistent or progressive focal neurological deficits despite an unrevealing stroke evaluation, clinicians should consider spinal pathology and pursue timely spinal imaging and neurosurgical consultation. Early recognition and surgical decompression may prevent permanent neurological deficits and significantly improve functional outcomes.

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