Acute monoplegia with Incontinence

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**Recommended Citation**

S, Dr. Ruthvik and P, Dr. Shastara (2021) "Acute monoplegia with Incontinence," *Digital Journal of Clinical Medicine*: Vol. 3: Iss. 1, Article 9.  
https://doi.org/10.55691/2582-3868.1087

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**Clinical History:**

A 52-year-old male patient developed insidious onset urinary incontinence 1 month ago. He has been on Foley’s catheter for the last 20 days for the same. The patient also complains of weakness of the right lower limb for 20 days. The weakness has been progressive and involves both the proximal and distal groups of muscles. There is dissociated sensory loss in the same limb as well. No history of similar complaints on the left lower limb or both the upper limbs.

The patient now complains of loose stools and burning micturition, associated with increased frequency since 3 days. No history of fever, trauma, neck pain or back pain, headache, and seizures. No history of neurological illness in the past or in the family. The patient is a known case of type 2 diabetes mellitus and hypertension for the last 15 years on medication. He is also a known case of diabetic chronic kidney disease on treatment.

**Examination and Investigations:**

*General physical examination* – conjunctival pallor present. Bilateral pitting pedal edema present.

Pulse – 84bpm

BP- 150/90mmHg

*CNS examination* –
Higher mental functions – Conscious, oriented to time, place, and person.

Cranial nerves- All cranial nerves are intact.

Motor system- Tone – spasticity in the right lower limb.

  Power- 0/5 in the Right lower limb.

  Deep tendon reflexes- brisk in the right lower limb.

  Plantar reflex- Extensor on the right side and flexor on the left side.

Sensory system- Dissociated sensory loss present on the right side, below the level of T4.

Pain and temperature are lost whereas touch and vibration sensations are preserved.

No significant motor or sensory changes in the other limbs.

No cerebellar signs.

No spinal abnormalities on examination.

**Per abdominal examination**– No abdominal distension. No local rise of temperature or rigidity was found. The abdomen is non-tender. No organomegaly or mass found.

**CVS examination**– S1 S2 heard. No murmurs heard.

**Respiratory system examination**– Bilateral air entry present. Bilateral NVBS heard. No added sounds were heard.

Complete haemogram revealed:

-Hb-9.6 gm%,

-TLC- 7390 cells/mm3.
Peripheral blood smear showed a picture of microcytic hypochromic anemia.

Urine routine showed 3+ albumin, 18-20 pus cells, and no casts.

LFT was normal.

His renal function tests showed blood urea of 83 mg/dl and serum creatinine of 4.0 and normal levels of serum electrolytes.

USG abdomen was done which revealed bilateral grade 1 medical renal disease and splenomegaly.

Urine culture- growth of E. coli.

MRI of the dorsal spine with whole spine screening revealed the following,

- Syringohydromyelia from C6-T9 with a mild expansion of the cord.
T1 weighted MRI images of the Sagittal view of the spine showing syringohydromyelia extending from C6-T9 (in white arrows).

**FINAL DIAGNOSIS:**

- Acute monoplegia and neurogenic bladder.
- Intramedullary pathology - Syringohydromyelia of the lower cervical and dorsal column, from C6-T9.
- Urinary tract infection

**DISCUSSION:**
Syringomyelia is a disorder of abnormal CSF circulation. A syrinx is a fluid-filled cavity that anatomically lies within the spinal cord parenchyma or the central canal[1]. Hydromyelia refers to a fluid-filled cavity within the spinal cord lined by ependymal, which likely results from a developmentally non-obliterated central canal. The two terms are often interchanged[2].

The natural history of patients is variable and unpredictable. It is interlaced with periods of stability and progression. The clinical course progresses over months to years. There is an early rapid deterioration that gradually slows down[3].

Epidemiological data on syringomyelia is limited. Some studies have found the prevalence of syringomyelia to be from 8.4/100,000 to 0.9/10,000 with an ethnic and geographic variation[4]. The majority of the patients present between ages 20 to 50 with a mean age of 30.

Etiology can be idiopathic, malformations like Chiari-I, post TB sequelae, post-traumatic, post-operative meningitis.

The presentation is highly variable. In most cases, patients complain of pain, muscle weakness, temperature insensitivity in the upper limb, spasticity or stiffness in lower limbs, progressive scoliosis, bladder incontinence.

MRI with and without contrast is the investigation of choice. It allows accurate visualization of the syrinx in both sagittal and axial planes. MRI easily reveals the location, size, and extent of the syrinx cavity.

All current treatment strategies are directed toward improving CSF flow dynamics.
Shunts are indicated for idiopathic syringomyelia. Most commonly used is the syringosubarachnoid shunt (SSAS). If this fails syringoperitoneal shunt (SPS) may be used. In most cases, shunts are not favored and are used as a last resort because of high complication and failure rates.

Spinal intramedullary tumors such as hemangioblastoma, ependymoma, gliomas are the differential diagnoses. Most of these intramedullary tumors will enhance on contrast MRI (a syrinx does not enhance on contrast MRI)[5].

Prognosis depends on the etiology, the degree of neurological deficit, and the site and size of the syrinx cavity. Syrinx diameter of more than 5 mm and associated edema predict a rapid deterioration. However, early surgery minimizes deficits and has better outcomes[6].

ACKNOWLEDGEMENTS: None

REFERENCES:


