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CASE REPORT

NEUROPATHIC PAIN IN SYRINGOMYELIA ASSOCIATED WITH ADULT CHIARI MALFORMATION TYPE 1: A CASE REPORT AND COMPREHENSIVE REVIEW OF CLINICAL MANAGEMENT

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Abstract

Introduction: Syringomyelia is an uncommon chronic spinal cord disorder characterized by the formation of a fluid-filled cavity (syrinx) within the neural parenchyma. This condition leads to progressive neurological impairment and severe neuropathic pain. This case report discusses the clinical presentation and comprehensive management of a patient with this condition.

Case Presentation: A 39-year-old female presented with a two-year history of severe burning pain, sensory loss in a "cape-like" distribution, and progressive weakness of the hands. Magnetic Resonance Imaging (MRI) of the cervical and thoracic spine demonstrated a prominent, hyperintense cervicothoracic syrinx (C2–T2) secondary to adult Chiari malformation type 1 (CM1).

Discussion: nitial gabapentin and amitriptyline yielded only partial pain control. Due to progressive lower motor neuron decline and refractory neuropathic pain, surgical intervention was indicated. Although foramen magnum decompression (FMD) remains the primary standard of care for restoring craniocervical CSF flow, a targeted syringo-subarachnoid shunting (SSS) procedure at the T1 level was used as a secondary rescue intervention to achieve rapid mechanical decompression of the massive, tense syrinx. Postoperatively, the patient demonstrated significant neurological improvement, with the Numeric Pain Rating Scale (NPRS) score decreasing from 7–8 to 3–4, alongside gradual motor recovery.

Conclusions: Neuropathic pain in syringomyelia is complex and requires a multimodal, multidisciplinary approach. While FMD is the primary gold standard for Chiari I–associated syringomyelia, SSS represents a viable secondary option for rapid mechanical decompression in cases presenting with giant, highly symptomatic syrinxes and progressive motor decline, despite carrying higher long-term revision and complication risks.

Keywords: Chiari Malformation, Chronic Pain, Neuropathic Pain, Syringomyelia, Syringo-Subarachnoid Shunt

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Introduction

Syringomyelia is a chronic neurological condition characterized by the development of a fluid-filled cyst within the spinal cord.[1] This cyst is medically referred to as a syrinx. Over time, the syrinx can enlarge and elongate. This process

compresses the surrounding nerve tissue. Consequently, patients experience spinal cord damage that triggers pain, weakness, and stiffness.[1] This disease is considered rare in daily clinical practice.[2] However, the impact on the patient's quality of life is significant.[2]

Epidemiologically, the prevalence of syringomyelia varies globally. In Western countries, the prevalence is recorded at approximately 8.4 per 100,000 population.[2] Meanwhile, a comprehensive nationwide epidemiological survey conducted in Japan demonstrated a lower ambulatory prevalence of 1.94 per 100,000.[1] In Indonesia, national data regarding the prevalence of this disease remains extremely limited.[3] While its progression varies, syringomyelia is characterized by a peak symptomatic onset in young adulthood, typically presenting between the third and fourth decades of life.[1,3] Although historically considered rare, the widespread availability of high-resolution Magnetic Resonance Imaging (MRI) has significantly increased the detection and diagnosis of both symptomatic and incidental cases in recent decades.[4]

The causes of syringomyelia can be divided into two main categories: congenital and acquired.[2] Chiari malformation type I (CM-1) is the cause most frequently associated (approximately 65% of CM-1 cases) with this condition.[1] In CM-1, brain tissue (cerebellar tonsils) protrudes into the spinal canal through the foramen magnum.[5] This obstructs the normal flow of cerebrospinal fluid (CSF).[1] Other types of syringomyelia can be caused by spinal cord trauma (1-9% of spinal cord injury patients), intramedullary tumors, or inflammation such as arachnoiditis.[6-8]

Neuropathic pain is one of the most difficult symptoms to manage in syringomyelia patients.[9] This pain arises due to direct damage to the somatosensory pathways in the spinal cord.[10] Patients often describe the pain as a burning, stabbing, or electric shock-like sensation.[3] This pain tends to be chronic and often does not respond well to standard pain medications.[11] Therefore, a management strategy involving pharmacological approaches, rehabilitation, and surgical intervention is necessary.[1]

The purpose of this case report is to present the clinical journey of a patient with syringomyelia experiencing refractory neuropathic pain. The authors will also discuss recent literature regarding the pathophysiological mechanisms of pain in syringomyelia. Furthermore, this report will review the effectiveness of various currently available treatment modalities. A deep understanding of this condition is vital for clinicians. This aims to ensure patients receive early diagnosis and appropriate intervention to prevent permanent neurological deficits.[4]

This report also aims to identify knowledge gaps in the management of syringomyelia in Indonesia. Limited access to certain neuropathic medications and advanced neurosurgical facilities remains a challenge in several regions.[3] By reviewing this case, it is hoped to provide practical guidance for neurologists facing similar cases in the future.

Case Presentation

A 39-year-old female patient presented to the neurology clinic with a primary complaint of severe numbness. In addition to numbness, the patient complained of burning and tingling in both arms and shoulders. These symptoms had persisted for the past 2 years and were slowly worsening. The patient described her pain as constant and very intense. On the Numeric Pain Rating Scale (NPRS), the patient reported a score of 7-8 out of 10. The pain was occasionally accompanied by shooting sensations radiating to the fingertips.

The patient also reported progressive weakness of the hand muscles. She frequently had difficulty holding small objects and often dropped items unintentionally. No bowel or bladder dysfunction was found. The patient's medical history showed no hypertension, diabetes, or malignancy. Family history also did not show similar neurological disorders. The patient is a housewife and is right-hand dominant.

A thorough neurological examination was performed to determine the site of nerve damage. The examination results showed bilateral atrophy of the intrinsic hand muscles. Muscle strength in the upper extremities was rated as grade 4/5 based on the Medical Research Council (MRC) scale. Sensory examination showed decreased pinprick and thermal sensation in the shoulder and arm areas. This distribution of sensory loss formed a characteristic "cape-like" pattern.[12] Interestingly, vibration sensation and proprioception remained well-preserved. This phenomenon, known as sensory dissociation, indicates damage to the spinal cord's central portion.[13] Deep tendon reflexes in the arms were brisk, but pathological reflexes such as the Babinski sign were absent.

Based on the progressive lower motor neuron deficits and typical sensory dissociation, the patient was scheduled for an MRI of the cervical and upper thoracic spinal cord. The sagittal T2-weighted MRI revealed a caudal descent of the cerebellar tonsils by approximately 6 mm below the foramen magnum, confirming the diagnosis of adult Chiari malformation type 1 (CM1).[6] Additionally, a prominent hyperintense fusiform intramedullary syrinx was observed extending from the C2 to the T2 vertebral levels (Figure 1), expanding the surrounding neural parenchyma. No other external compressive masses or space-occupying lesions were identified. Routine laboratory tests, including a complete blood count, metabolic panel, and kidney function tests, were within normal limits, ruling out systemic inflammation or nutritional deficiency as the primary causes of symptoms.[13]

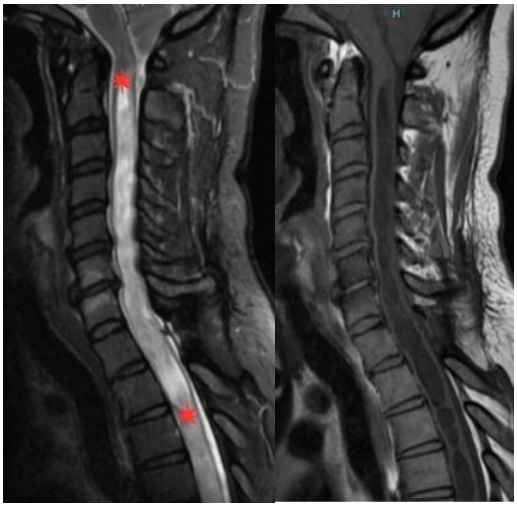
Initial management was carried out using pharmacological and physical rehabilitation approaches. Drug therapy included oral gabapentin. The gabapentin dose was gradually titrated up to 900 mg per day. This therapy provided only partial pain reduction. To optimize pain control and address the patient's sleep disturbances, amitriptyline was added at a dose of 10–25 mg before bed. The patient was also referred to a specialized physical therapy program focused on upper-arm muscle strengthening, coordination, and

functional mobility.[2] During therapy, the patient was educated on joint protection to prevent injury due to loss of pain sensation.[11]

Despite intensive medical therapy, the patient's condition continued to show a decline in muscle strength over the following six months. The neuropathic pain also became increasingly severe and uncontrolled. After a multidisciplinary discussion with the neurosurgery team, it was decided that the patient required surgical intervention. Although FMD is the primary, gold-standard surgical paradigm for correcting craniocervical CSF dynamics in CM1, the clinical team elected to perform a syringo-subarachnoid shunting (SSS) procedure as a highly selective rescue intervention.[14] This approach was chosen because of the massive, highly tense nature of the cervicothoracic syrinx (C2–T2) and the rapid, progressive loss of bilateral hand motor function, prioritizing immediate, direct mechanical deflation of the cord parenchyma over gradual decompression.[15]

Under general anesthesia, the patient was positioned prone with the head stabilized in a skull clamp. Continuous motor- and somatosensory-evoked potentials (MEPs/SSEPs) were maintained throughout. To access the syrinx while avoiding direct mechanical trauma to the cervical enlargement, a localized C7–T1 laminectomy was performed. Under microscopic magnification, a midline durotomy was performed, and the arachnoid membrane was opened to expose the dorsal spinal cord. A 2 mm longitudinal myelotomy was performed along the posterior median sulcus at the T1 level. A soft silicone T-tube catheter (internal diameter 1.32 mm, external diameter 1.5 mm) was inserted, with the proximal arm directed rostrally into the syrinx cavity and the distal arm positioned in the anterolateral subarachnoid space. This anterolateral placement was selected to reduce the risk of trabecular arachnoid scarring and shunt occlusion. The catheter was secured to the pia mater at the myelotomy margin with a single 6-0 polypropylene suture to prevent migration. Watertight dural closure was achieved with running 4-0 nylon sutures and dural sealant, and an intraoperative Valsalva maneuver confirmed satisfactory closure up to 30 cm H_2O before layered wound closure.

Post-surgery, the patient showed significant clinical improvement. Pain intensity decreased noticeably to a score of 3–4 on the NPRS scale. Additionally, the patient's muscle strength gradually improved over the next three months. At the six-month follow-up after surgery, the patient's neurological condition remained stable. The patient reported an increase in quality of life and independence in daily activities. Currently, the patient continues to take a maintenance dose of pregabalin 75 mg b.i.d., amitriptyline 25 mg once daily, and tramadol for breakthrough pain. The patient has provided informed consent for the publication of this medical data.



(A) (B)

Figure 1. Sagittal MRI of the cervical and upper thoracic spine. (A) T2-weighted image demonstrates a prominent, hyperintense intramedullary syrinx extending from the C2 to the T2 vertebral levels (delineated by red asterisks). (B) The T1-weighted image shows the corresponding hypointense signal within the spinal cord, confirming the presence of an extensive fluid-filled cavity consistent with syringomyelia

Discussion

Syringomyelia in this patient is secondary to adult Chiari malformation type 1 (CM1), which involves a profound disruption of craniospinal CSF dynamics.[1] In CM1, underdevelopment of the posterior fossa leads to crowding of the cerebellum and the caudal displacement of the cerebellar tonsils through the foramen magnum.[6] With each cardiac cycle, the displaced tonsils act as a piston, creating a 'water-hammer' effect that drives CSF downward and blocks the normal pulsatile flow between the intracranial and spinal subarachnoid spaces.[4,6] This pressure gradient forces fluid into the spinal cord parenchyma, culminating in the formation and progressive expansion of a central syrinx.[4] Neuropathic pain arises directly from this intraparenchymal expansion, which severs the crossing fibers of the spinothalamic tract at the anterior white commissure and compresses the sensory pathways in the dorsal horn.[12,16] Beyond mechanical damage, functional changes occur at the cellular level, with nerve damage triggering glial activation and the release of pro-inflammatory cytokines, thereby driving central sensitization and refractory chronic pain.[17,18]

Managing neuropathic pain in syringomyelia requires a systematic, evidence-based approach.[19] Tricyclic antidepressants (TCAs) and gabapentinoids represent the cornerstones of pharmacological therapy.[3,19] These strategies are closely aligned with the international recommendations of the International Association for the Study of Pain (IASP) Neuropathic Pain Special Interest Group (NeuPSIG) and the National Institute for Health and Care Excellence (NICE) Clinical Guideline CG173.[20,21] According to the landmark NeuPSIG systematic review and meta-analysis, TCAs, gabapentin, and pregabalin are strongly recommended as first-line agents.[22] The combined Number Needed to Treat (NNT) for a 50% pain reduction was established at 3.6 (95% CI 3.0–4.4) for TCAs, 6.3 (95% CI 5.0–8.3) for gabapentin, and 7.7 (95% CI 6.5–9.4) for pregabalin.[22] In the updated NeuPSIG systematic review and meta-analysis evaluating 313 trials involving 48,789 adult patients, these strong first-line recommendations were reaffirmed, reporting an NNT of 4.6 (95% CI 3.2–7.7) for TCAs and 8.9 (95% CI 7.4–11.1) for $\alpha 2\delta$-ligands (gabapentinoids).[23] The upward

shift in NNT over time reflects modern methodological designs, larger sample sizes, and heightened placebo responses in clinical trials, rather than a decline in absolute clinical utility.[22,23]

In this case, the patient's initial pharmacological regimen consisted of oral gabapentin titrated to 900 mg daily. Based on NICE CG173, gabapentin titration should start at 300 mg daily (or 100 mg three times daily) and be gradually increased weekly to a standard therapeutic dose of 1200–1800 mg daily, up to a maximum of 3600 mg daily as tolerated.[20] The patient's sub-therapeutic dose of 900 mg/day yielded only partial pain relief, which is consistent with the established NNT of gabapentinoids, where more than half of treated patients fail to achieve a 50% pain reduction and instead experience limiting side effects.[24] To optimize pain control and address severe sleep disturbances, amitriptyline was added at a low dose of 10 to 25 mg before bed. Amitriptyline enhances descending monoaminergic pain-inhibition pathways by blocking the reuptake of serotonin and norepinephrine.[25] However, its clinical application is frequently restricted by anticholinergic side effects (e.g., dry mouth, somnolence, urinary retention, and constipation), carrying a Number Needed to Harm (NNH) of 13.4 in 2015 and 17.1 in 2025.[23,25]

Post-surgery, the patient was successfully switched to a maintenance regimen of pregabalin (75 mg b.i.d.), amitriptyline (25 mg once daily), and tramadol for breakthrough pain. Pregabalin possesses superior bioavailability, a more linear pharmacokinetic profile, and a favorable safety margin compared to gabapentin.[25] The standard pregabalin maintenance dose is initiated at 150 mg daily (divided as 75 mg twice daily) and can be titrated to a maximum of 600 mg daily.[22,23] Tramadol, a weak μ-opioid agonist with serotonin and noradrenaline reuptake inhibition, was reserved strictly for breakthrough pain.[22] Both NICE CG173 and NeuPSIG guidelines advise that tramadol should only be used as acute rescue therapy for breakthrough pain rather than a continuous long-term first-line treatment due to risks of tolerance, dependence, and central nervous system depression.[20,23]

Neurosurgical intervention is indicated for symptomatic patients demonstrating progressive neurological deficits, intractable pain, or an expanding syrinx volume on serial MRI.[26] In patients with CM1-associated syringomyelia, posterior fossa decompression–typically executed as foramen magnum decompression (FMD) with duraplasty–is universally recognized as the primary, first-line standard of care.[1] FMD directly addresses the causative pathology at the craniocervical junction, restoring physiological CSF pathways and facilitating long-term syrinx resolution in approximately 80% of cases.[26] Direct shunting procedures, such as syringo-subarachnoid shunting (SSS), do not correct the underlying craniocervical obstruction and are therefore not preferred as primary therapy.[1] International consensus and clinical guidelines relegate SSS to a secondary rescue option reserved for refractory cases or select clinical scenarios characterized by

massive, highly tense syrinxes and rapid, progressive lower motor neuron decline.[14]

The clinical decision to utilize SSS in this patient was guided by the need for immediate mechanical deflation of a giant cervicothoracic cavity to halt progressive anterior horn cell ischemia.[14] Comparative clinical data demonstrate that SSS can achieve a significantly shorter mean time to radiological syrinx collapse (1.8 weeks) compared to FMD (6.3 weeks).[28] Furthermore, in cases of massive syringomyelia where the syrinx width exceeds 70% of the spinal cord width, direct mechanical diversion has demonstrated rapid, highly pronounced motor recovery and immediate neuropathic pain relief.[28] Despite these immediate decompression benefits, SSS is associated with a significantly higher long-term failure and revision rate compared to FMD (10% to 28 %) for SSS versus approximately 9% to 10% for FMD.[15,28] Shunt failure is primarily driven by catheter migration, distal mechanical occlusion, and localized adhesive arachnoiditis around the shunt tube.[14] Consequently, FMD remains the preferred, evidence-based standard of care. In contrast, SSS must be strictly restricted to a rescue role in cases of rapid neurological decline where immediate structural decompression is clinically prioritized over long-term device patency.[1]

Non-pharmacological therapy plays a vital role in long-term recovery.[3] Physiotherapy aims to train residual muscle strength and improve coordination.[4] Neurodynamic techniques can help optimize neural tissue movement and improve blood flow to compressed nerves.[17] The use of modalities such as Transcutaneous Electrical Nerve Stimulation (TENS) and Short-Wave Diathermy (SWD) has been reported to provide additional benefits in reducing chronic pain for some patients in Indonesia.[2] Furthermore, education on lifestyle modification is essential. Patients are advised to avoid activities that suddenly increase intrathoracic pressure, such as heavy lifting or excessive straining (Valsalva maneuver), as this can worsen CSF flow and expand the syrinx.[11] Psychological support must also not be ignored. Chronic pain often leads to depression and social isolation.[3] Cognitive-behavioral therapy (CBT) and breathing-relaxation techniques can help patients develop healthy coping strategies for persistent pain.[3]

One rare but very destructive complication of syringomyelia is neuropathic arthropathy or Charcot joint. This condition results from loss of pain sensation and proprioception (joint position sense).[29] Consequently, the joint undergoes repeated microtrauma of which the patient is unaware.[30] This triggers progressive bone and joint destruction, often in the shoulder or elbow.[29] Clinicians must be alert for painless joint swelling in patients with syringomyelia, as early diagnosis is crucial to prevent permanent disability.[10]

Conclusion

This case report demonstrates that neuropathic pain in syringomyelia is a major clinical challenge. The clinical presentation of sensory dissociation with a "cape-like" pattern is a key indicator for performing an immediate spinal cord MRI.

Initial management using first-line medical therapy such as amitriptyline and gabapentinoids often yields limited results if clear structural abnormalities exist.

While FMD represents the primary standard of care for correcting the underlying pathophysiology of Chiari-associated syringomyelia, the utilization of a targeted syringo-subarachnoid shunt (SSS) at the T1 level served as an effective secondary rescue intervention in this specific patient. SSS provided immediate mechanical decompression of a massive, tense syrinx, successfully arresting acute lower motor neuron deterioration and significantly alleviating refractory neuropathic pain. Nonetheless, because of the elevated long-term risks of catheter migration, mechanical obstruction, and localized adhesive arachnoiditis associated with shunting devices, SSS must not be regarded as a primary standard of care. Long-term clinical success depends on vigilant, longitudinal follow-up, continuous multidisciplinary rehabilitation, and a comprehensive understanding of the structural and pharmacological complexities of spinal cord injury.

Therefore, every clinician needs to understand the pathophysiology of syringomyelia comprehensively. Collaboration among neurologists, neurosurgeons, and physical medicine and rehabilitation specialists is essential to achieve optimal clinical outcomes. The future of syringomyelia management is expected to involve more advanced neuromodulation modalities to address neuropathic pain that persists after surgical intervention.

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Conflict of Interest

The authors declared no conflict of interest.

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Consent

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