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Case Report

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Concurrent Guillain-Barré Syndrome and Thoracic Syringomyelia in a Pediatric Patient: A Case Report

Kailee Sowers[1], Nyah Manuel[1], Jack Ojile[1], Keerthivasan Ramesh[2] and Ali Jamal[2]*

[1]Saint Louis University School of Medicine, St. Louis, MO, USA

[2]Department of Pediatric Neurology, Saint Louis University School of Medicine, St. Louis, MO, USA

Abstract

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyneuropathy characterized by ascending weakness, areflexia, and risk of respiratory failure. Syringomyelia is the presence of a fluid-filled cavity, or syrinx, within the spinal cord that may occur due to genetic or acquired causes. We report the case of an 8-year-old boy with an atypical presentation of GBS, characterized by fluctuating ataxia and lower extremity pain. The diagnosis of GBS was supported by cerebrospinal fluid analysis demonstrating albuminocytologic dissociation and magnetic resonance imaging (MRI) findings of cauda equina nerve root enhancement. MRI also revealed a thoracic syrinx extending from T5-T10/11, prompting neurosurgical evaluation. The patient was managed conservatively and treated with a three-day course of intravenous immunoglobulin, with subsequent clinical improvement and discharge home. He returned to baseline function and resumed school attendance three months after discharge. The coexistence of GBS and syringomyelia is rare, and the relationship between these conditions remains uncertain. This case highlights an unusual presentation of GBS associated with thoracic syringomyelia and raises the possibility that altered cerebrospinal fluid dynamics during acute inflammatory neuropathy may contribute to syrinx formation.

Additional reports of similar cases may help clarify the association, clinical course, and outcomes of patients with concurrent GBS and syringomyelia.

Keywords

Guillain-Barré syndrome, Syringomyelia, Syrinx

Introduction

Guillain-Barré Syndrome (GBS) is an acute immune-mediated peripheral neuropathy characterized by progressive weakness, diminished or absent deep tendon reflexes, and varying degrees of sensory involvement [1]. Symptoms typically develop within four weeks of an immunological trigger and may progress to life-threatening complications including respiratory failure requiring ventilatory support [2]. Diagnosis is primarily clinical and is supported by ancillary studies such as nerve conduction studies, cerebrospinal fluid (CSF) analysis, and magnetic resonance imaging (MRI). Treatment consists of immunotherapy with intravenous immunoglobulin (IVIG) or plasmapheresis, along with supportive management [3].

A syrinx is a fluid-filled cavity within the spinal cord that can result from congenital abnormalities, including Chiari malformation or tethered cord syndrome, or develop secondary to acquired conditions such as trauma, infection or neoplasm. Clinical manifestations vary depending on the size and location of the syrinx and may include sensory disturbances, weakness, pain, and gait abnormalities [4]. Diagnosis is established through MRI and management ranges from observation to surgical intervention based on symptom severity and progression [5].

Although both GBS and syringomyelia can present with sensorimotor deficits, their simultaneous occurrence is rarely reported, and the relationship between the two conditions remains poorly understood. Distinguishing between clinical findings caused by peripheral nerve dysfunction and spinal cord pathology can present a diagnostic challenge. Here,

***Corresponding author:** Saint Louis University School of Medicine, 300 S. Grand Blvd. Apt. 613, St. Louis, MO, 63103, USA

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we describe the case of an 8-year-old boy with concurrent GBS and thoracic syringomyelia, highlighting an unusual presentation and the clinical considerations involved in diagnosis and management.

Case Presentation

An 8-year-old male with no significant past medical or surgical history presented to the emergency department with a two-week history of fluctuating bilateral leg pain, weakness, difficulty walking, and fatigue. His symptoms began with a low-grade fever and right thigh pain that improved with ibuprofen. Days later, the pain recurred and progressed to involve both lower extremities, his back, and neck. His mother noted progressive instability, frequent falls, and a slow, wide-based gait. He initially presented to an outside hospital and was diagnosed with a hamstring injury; however, his symptoms persisted with fluctuating periods of improvement and worsening.

Due to persistent and progressive symptoms, he presented to our emergency department for further evaluation. At that time, he reported bilateral lower extremity paresthesias but remained able to ambulate independently.

Neurologic examination revealed mild symmetric upper extremity weakness (4+/5) and lower extremity weakness (4/5). Deep tendon reflexes were absent at the bilateral patellar and Achilles tendons but preserved in the upper extremities. Sensory examination revealed decreased sensation to light touch below the knees bilaterally. He demonstrated a slow, wide-based gait with compensatory knee flexion and arm abduction to maintain balance.

MRI of the spine with and without contrast demonstrated diffuse enhancement and thickening of the dorsal and ventral nerve roots along the cauda equina (Figures 1 and 2), which raised concern for GBS. However, imaging also revealed a thoracic syrinx extending from T5 to T10/11 (Figures 3a, 3b, and 4), prompting neurosurgical evaluation. MRI brain was obtained to assess for associated structural abnormalities and demonstrated leptomeningeal enhancement surrounding the upper cervical spinal cord and medulla.

Lumbar puncture revealed albuminocytologic dissociation with elevated CSF protein of 223 mg/dL, normal cell count (1 cell/mm³), and normal glucose (58 mg/dL). CSF viral studies were negative. Based on the clinical presentation, CSF findings, and MRI evidence of cauda equina enhancement, the patient was diagnosed with GBS and treated with IVIG at a total dose of 2 g/kg divided over three days.

Neurosurgery recommended conservative management of the thoracic syrinx with outpatient follow-up, as there were no clear findings attributable to the syrinx. During hospitalization, he had intermittent episodes of urinary incontinence. His mother reported baseline nighttime urinary incontinence requiring pull-ups prior to admission. Given the concern for possible neurologic involvement, urinary symptoms were monitored to distinguish baseline enuresis from new bladder dysfunction or functional limitations related to lower extremity weakness. Following completion of IVIG, the patient demonstrated improvement in balance, posture, and mental status. He was discharged home with outpatient physical therapy and follow-up with neurology and neurosurgery.

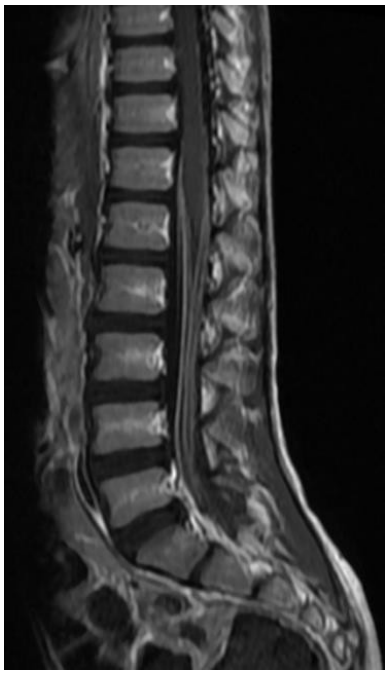

Figure 1: MRI Spine Sagittal T1 Flair with contrast showing cauda equina nerve root enhancement.

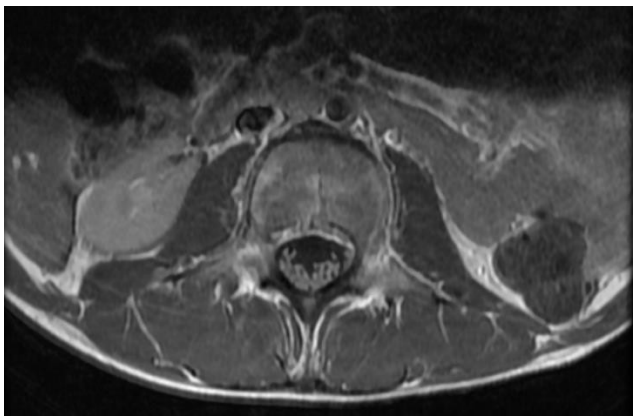

Figure 2: MRI Spine Axial T1 with contrast showing cauda equina nerve root enhancement.

At follow-up one month after discharge, he was ambulating without a walker and continued to demonstrate improvement in coordination and gait. Mild bilateral lower extremity weakness and absent lower extremity reflexes persisted. At a subsequent follow-up two months later, strength had returned to 5/5 throughout, although lower extremity reflexes remained absent. He was cleared to return to school. Given his continued clinical improvement and absence of progressive neurologic deficits attributable to the syrinx, neurosurgical intervention was not pursued, and continued clinical monitoring was recommended.

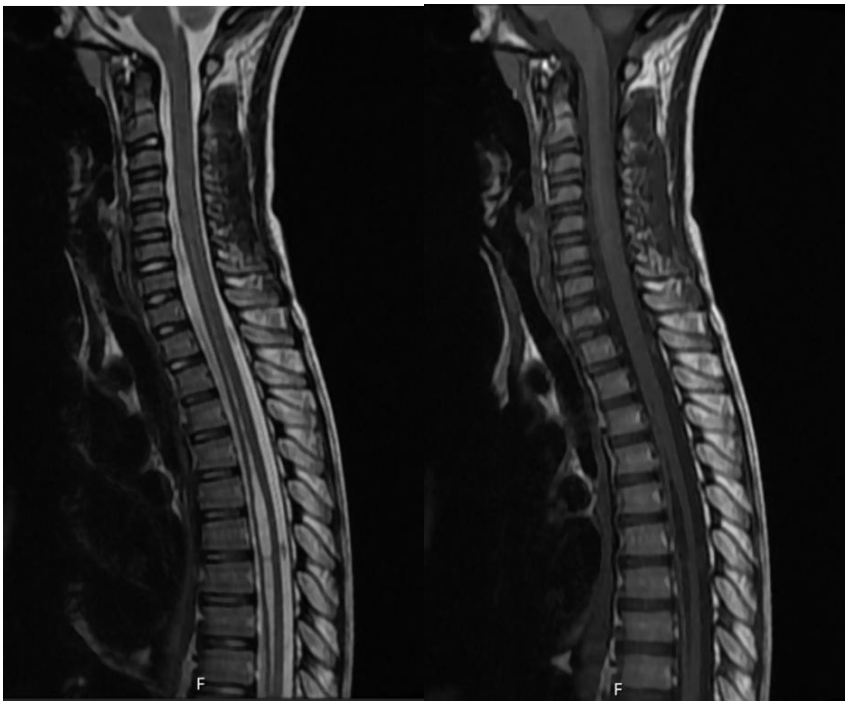

Figure 3: (a): MRI Spine Sagittal T2 FRFSE of thoracic syrinx; (b): Corresponding T1 Flair of thoracic syrinx.

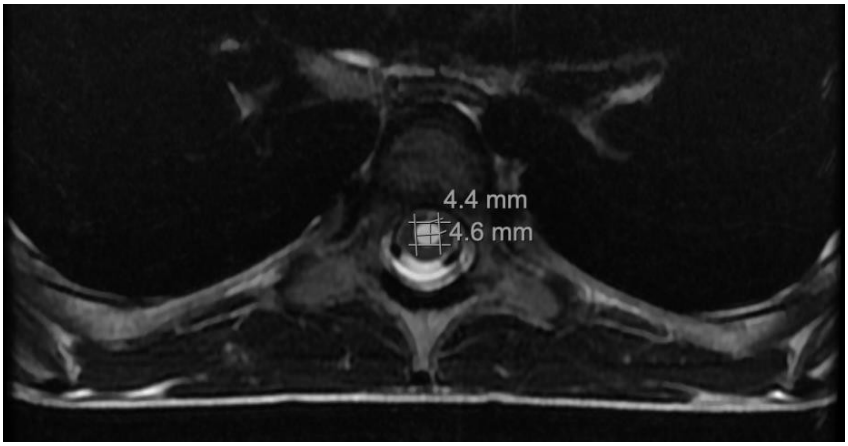

Figure 4: MRI Spine axial view of thoracic syrinx with measurements.

Discussion

This patient presented with findings consistent with GBS, including progressive, ascending sensorimotor deficits, areflexia, albuminocytologic dissociation, and cauda equina nerve root enhancement on MRI. However, his presentation was uniquely notable for fluctuating symptoms and unexpected discovery of a thoracic syrinx, creating a diagnostic consideration regarding the contribution of spinal cord involvement in his clinical course.

Although GBS classically presents with progressive symmetric ascending weakness, it is known to have variability in its presentation. Studies have shown that as many as 23% of pediatric patients display asymmetric weakness, and paraparetic GBS is a milder form that presents with lower extremity weakness without further ascension [6,7]. There have been numerous reports of patients with rarer presentations including pure sensory involvement, pharyngeal-cervical-brachial variants, and respiratory failure as the presenting symptom [8-10]. In this patient, the waxing and waning nature of symptoms initially complicated recognition of the underlying neuropathy. The additional MRI finding of leptomeningeal enhancement prompted consideration of broader infectious involvement; however, the clinical presentation, cerebrospinal fluid (CSF) abnormalities, and characteristic cauda equina enhancement remained most consistent with GBS.

The significance of the thoracic syrinx in this patient remains uncertain. His history and presentation lack common causes of acquired syringomyelia such as trauma, arachnoid

cyst, or meningitis [5]. Given the presence of the syrinx, the patient was evaluated for clinical features suggestive of spinal cord involvement; however, no progressive focal deficits or examination findings clearly attributable to the syrinx were identified during hospitalization. The patient had a history of primary nocturnal enuresis predating presentation. Given its longstanding nature, absence of daytime urinary symptoms, and lack of progressive change, this was considered unlikely to represent spinal cord dysfunction attributable to the syrinx. Episodes of incontinence during hospitalization were thought to be more consistent with impaired mobility during the acute illness rather than new neurogenic bladder dysfunction.

To our knowledge, only two published cases have described concurrent GBS and syringomyelia. The first was a 67-year-old male initially hospitalized for classic GBS and later rehospitalized for sensory deficits, spasticity, and urinary incontinence related to a syrinx on cervical MRI [11]. The second case regarded a 6-year-old female presenting with progressive sensory and motor loss of bilateral lower extremities with MRI showing C2-T3 syringomyelia [12]. Both patients recovered without complications related to either syrinx or GBS.

The relationship between these conditions remains unclear. It is possible that the two processes occurred independently or shared a post-infectious inflammatory mechanism contributing to their coexistence. There have been cases of GBS presenting with signs of increased intracranial pressure, thought to be due to increased CSF protein [7]. One proposed explanation is that inflammatory changes associated with GBS, including elevated CSF protein, may alter CSF dynamics and contribute to syrinx formation; however further cases are needed to evaluate this hypothesis.

The patient was successfully treated with a three-day course of IVIG and demonstrated improvement in gait stability, coordination and strength. Three months after discharge, he had returned to baseline strength although lower extremity reflexes remained absent, which can persist up to months after GBS infection [3]. Conservative management of the thoracic syrinx was pursued due to absence of progressive focal deficits attributable to the lesion. Continued clinical monitoring remains important and repeat imaging or neurosurgical reassessment would be warranted if new or progressive symptoms develop [13].

This case highlights the importance of considering alternative or concurrent neurologic pathology when imaging reveals unexpected findings during evaluation of GBS. Further documentation of similar cases may help clarify the relationship between GBS and syringomyelia and guide management strategies for patients with both conditions.

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